(1) Structure search in Registry database with Registry hit structures searched in CAplus to obtain references.


```
=> file hcaplus
FILE 'HCAPLUS' ENTERED AT 14:56:38 ON 03 FEB 2003
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2003 AMERICAN CHEMICAL SOCIETY (ACS)
```

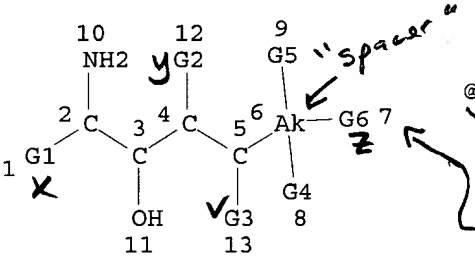


```
FILE COVERS 1907 - 3 Feb 2003  VOL 138 ISS 6
FILE LAST UPDATED: 2 Feb 2003  (20030202/ED)

 This file contains CAS Registry Numbers for easy and accurate
 substance identification.
```

"display query"

```
=> d que l17
L8                  STR
```



generic alkyl group, open - can be OH or ester

open - allows for H, alkyl, acyl.

CH2-O @14 15 — CH2-N @16 17 — part of G1 (X)

open - allows W to be anything including H

```
x  VAR G1=H/CH3/14/16
y  VAR G2=H/O
z  VAR G3=H/O
   VAR G4=H/OH
   VAR G5=H/OH
   VAR G6=H/C/N/O
NODE ATTRIBUTES:
CONNECT IS E3  RC AT  2
CONNECT IS E3  RC AT  3
CONNECT IS E3  RC AT  4
CONNECT IS E3  RC AT  5
CONNECT IS X4  RC AT  6
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED
ECOUNT  IS M1-X20 C  AT  6
```

allows generic alkyl (Ak) @ 6 to be alkyl ($G_4 = G_5 = H$), hydroxy alkyl (one of G_4 & G_5 = OH) or dihydroxy alkyl (both G_4 & G_5 = OH).

← limits generic Ak @ 6 to having 1–20 carbons.

```
GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS  17

STEREO ATTRIBUTES: NONE
L9                SCR 405
L10               SCR 1146
```

— search would not run to completion — structure too complex - without using screens to narrow portion of

Searched by Thom Larson, STIC, 308-7309

Registry database searched.

(see back)

search File /=Registry for structures

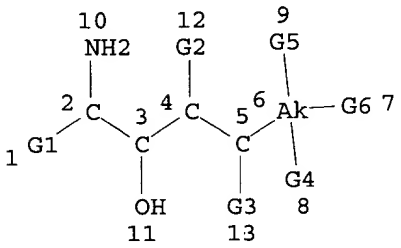
```
L11             SCR 1700
L12             SCR 1568
L13             SCR 2043
L14 (       909)SEA FILE=REGISTRY SSS FUL L8 AND L9 AND L10 AND L11 AND L12
                NOT L13
L15 (      8624)SEA FILE=HCAPLUS ABB=ON  PLU=ON  L14
L16 (      1029)SEA FILE=HCAPLUS ABB=ON  PLU=ON  FUMONISIN/CT OR FUMONISINS/CT
                OR FUMONISIN B1/CT
L17           2 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L16 AND L15
```

— polymers.

— removes polymers from answer set.

search CAplus with structures from Registry (L14) and key words in Controlled Term (CT) field.

```
=> d que l27
L18             STR
```



CH2·O @14 15

CH2·N @16 17

same structure search as before. It was assigned new L#s because I saved the search and reactivated it the next day. That is also where the parenthesis come from.

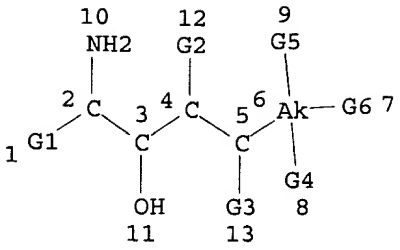
```
VAR G1=H/CH3/14/16
VAR G2=H/O
VAR G3=H/O
VAR G4=H/OH
VAR G5=H/OH
VAR G6=H/C/N/O
NODE ATTRIBUTES:
CONNECT IS E3  RC AT   2
CONNECT IS E3  RC AT   3
CONNECT IS E3  RC AT   4
CONNECT IS E3  RC AT   5
CONNECT IS X4  RC AT   6
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED
ECOUNT  IS M1-X20 C  AT   6

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS  17

STEREO ATTRIBUTES: NONE
L19             SCR 405
L20             SCR 1146
L21             SCR 1700
L22             SCR 1568
L23             SCR 2043
L24 (       909)SEA FILE=REGISTRY SSS FUL L18 AND L19 AND L20 AND L21 AND L22
                NOT L23
L25 (      8624)SEA FILE=HCAPLUS ABB=ON  PLU=ON  L24
L26 (      1419)SEA FILE=HCAPLUS ABB=ON  PLU=ON  FUMONISIN
L27           3 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L25 AND L26
```

← searched free text

```
=> d que l39
L28             STR
```



CH2·O @14 15

CH2·N @16 17

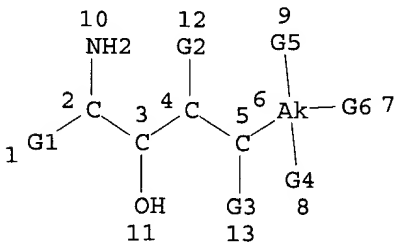
same structure search

VAR G1=H/CH3/14/16
VAR G2=H/O
VAR G3=H/O
VAR G4=H/OH
VAR G5=H/OH
VAR G6=H/C/N/O
NODE ATTRIBUTES:
CONNECT IS E3 RC AT 2
CONNECT IS E3 RC AT 3
CONNECT IS E3 RC AT 4
CONNECT IS E3 RC AT 5
CONNECT IS X4 RC AT 6
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED
ECOUNT IS M1-X20 C AT 6

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS 17

STEREO ATTRIBUTES: NONE
L29 SCR 405
L30 SCR 1146
L31 SCR 1700
L32 SCR 1568
L33 SCR 2043
L34 (909) SEA FILE=REGISTRY SSS FUL L28 AND L29 AND L30 AND L31 AND L32 NOT L33
L35 (431) SEA FILE=HCAPLUS ABB=ON PLU=ON L34 (L) THU/RL ≡ Therapeutic Role
L36 (209988) SEA FILE=HCAPLUS ABB=ON PLU=ON NEOPLASM+NT/CT OR NEOPLASMS+NT/CT
L37 (152548) SEA FILE=HCAPLUS ABB=ON PLU=ON ANTITUMOR AGENTS+NT1,PFT/CT
L38 (21) SEA FILE=HCAPLUS ABB=ON PLU=ON L35 AND L36
L39 14 SEA FILE=HCAPLUS ABB=ON PLU=ON L38 AND L37

=> d que l49
L40 STR



CH2·O @14 15

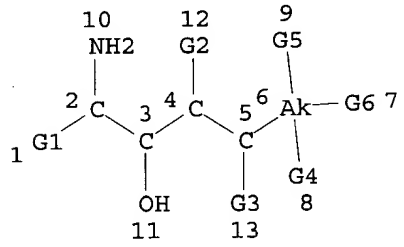
CH2·N @16 17

```
VAR G1=H/CH3/14/16
VAR G2=H/O
VAR G3=H/O
VAR G4=H/OH
VAR G5=H/OH
VAR G6=H/C/N/O
NODE ATTRIBUTES:
CONNECT IS E3  RC AT   2
CONNECT IS E3  RC AT   3
CONNECT IS E3  RC AT   4
CONNECT IS E3  RC AT   5
CONNECT IS X4  RC AT   6
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED
ECOUNT  IS M1-X20 C  AT   6

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS  17

STEREO ATTRIBUTES: NONE
L41              SCR 405
L42              SCR 1146
L43              SCR 1700
L44              SCR 1568
L45              SCR 2043
L46 (       909) SEA FILE=REGISTRY SSS FUL L40 AND L41 AND L42 AND L43 AND L44
                 NOT L45
L47 (      8624) SEA FILE=HCAPLUS ABB=ON  PLU=ON  L46
L48 (       304) SEA FILE=HCAPLUS ABB=ON  PLU=ON  "GANGLIOSIDOSIS (L) TAY-SACHS
                 DISEASE"+NT,PFT/CT
L49            0 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L47 AND L48


=> d que l59
L50              STR
```



CH2·O
@14 15

CH2·N
@16 17

```
VAR G1=H/CH3/14/16
VAR G2=H/O
VAR G3=H/O
VAR G4=H/OH
VAR G5=H/OH
VAR G6=H/C/N/O
NODE ATTRIBUTES:
CONNECT IS E3  RC AT   2
CONNECT IS E3  RC AT   3
CONNECT IS E3  RC AT   4
CONNECT IS E3  RC AT   5
CONNECT IS X4  RC AT   6
```

```
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED
ECOUNT  IS M1-X20 C  AT   6

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS  17

STEREO ATTRIBUTES: NONE
L51             SCR 405
L52             SCR 1146
L53             SCR 1700
L54             SCR 1568
L55             SCR 2043
L56 (       909)SEA FILE=REGISTRY SSS FUL L50 AND L51 AND L52 AND L53 AND L54
                NOT L55
L57 (      8624)SEA FILE=HCAPLUS ABB=ON  PLU=ON  L56
L58 (       490)SEA FILE=HCAPLUS ABB=ON  PLU=ON  NIEMANN-PICK DISEASE+NT,PFT/CT

L59           1 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L57 AND L58


=> d que l62
L60 (      1029)SEA FILE=HCAPLUS ABB=ON  PLU=ON  FUMONISIN/CT OR FUMONISINS/CT
                OR FUMONISIN B1/CT
L61 (       304)SEA FILE=HCAPLUS ABB=ON  PLU=ON  "GANGLIOSIDOSIS (L) TAY-SACHS
                DISEASE"+NT,PFT/CT
L62           1 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L60 AND L61


=> d que l65
L63 (      1029)SEA FILE=HCAPLUS ABB=ON  PLU=ON  FUMONISIN/CT OR FUMONISINS/CT
                OR FUMONISIN B1/CT
L64 (       490)SEA FILE=HCAPLUS ABB=ON  PLU=ON  NIEMANN-PICK DISEASE+NT,PFT/CT

L65           1 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L63 AND L64


=> d que l68
L66 (      1419)SEA FILE=HCAPLUS ABB=ON  PLU=ON  FUMONISIN
L67 (       304)SEA FILE=HCAPLUS ABB=ON  PLU=ON  "GANGLIOSIDOSIS (L) TAY-SACHS
                DISEASE"+NT,PFT/CT
L68           1 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L66 AND L67


=> d que l71
L69 (      1419)SEA FILE=HCAPLUS ABB=ON  PLU=ON  FUMONISIN
L70 (       490)SEA FILE=HCAPLUS ABB=ON  PLU=ON  NIEMANN-PICK DISEASE+NT,PFT/CT

L71           1 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L69 AND L70


=> s L17 or L27 or L39 or L49 or L59 or L62 or L65 or L68 or L71
L72          19 L17 OR L27 OR L39 OR L49 OR L59 OR L62 OR L65 OR L68 OR L71

=> D IBIB ABS HITSTR L72 1-19

L72  ANSWER 1 OF 19  HCAPLUS  COPYRIGHT 2003 ACS
ACCESSION NUMBER:         2003:5976  HCAPLUS
DOCUMENT NUMBER:          138:66707
```

Key terms only - no structure

} Combine all answer sets

Display Bibliographic info + abstract and hit structures.

TITLE: Neuraminic acid derivatives for use as Siglec inhibitors
INVENTOR(S): Kelm, Sorge; Brossmer, Reinhard
PATENT ASSIGNEE(S): Germany
SOURCE: PCT Int. Appl., 27 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003000709	A2	20030103	WO 2002-EP6277	20020607

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

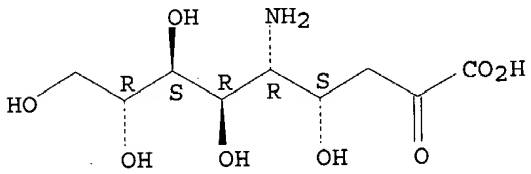
PRIORITY APPLN. INFO.: DE 2001-10129332 A 20010619
DE 2002-10216310 A 20020412

AB The invention discloses Siglec inhibitors that have an increased affinity for the receptor mol. The Siglec inhibitors of the invention are preferably selective for a given Siglec mol. The invention further discloses a method for producing Siglec inhibitors, as well as a method for increasing the binding selectivity for a given Siglec mol. The invention also discloses pharmaceutical compns. that contain the Siglec inhibitors and medical indications for the Siglec inhibitors. Prepn. of methyl-.alpha.-5-N-acetyl-9-N-(biphenyl-4-carbonyl)amino-9-desoxyneuraminic acid is described,.

IT **114-04-5D**, Neuraminic acid, derivs.
RL: PAC (Pharmacological activity); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(neuraminic acid derivs. as Siglec inhibitors, and therapeutic use)

RN 114-04-5 HCAPLUS
CN Neuraminic acid (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L72 ANSWER 2 OF 19 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:540258 HCAPLUS
DOCUMENT NUMBER: 137:109267
TITLE: Preparation of benzoxepinopyridines as HMG-CoA reductase inhibitors
INVENTOR(S): Robl, Jeffrey A.; Chen, Bang-chi; Sun, Chong-qing
PATENT ASSIGNEE(S): USA

SOURCE: U.S. Pat. Appl. Publ., 42 pp., Cont.-in-part of U.S. Ser. No. 875,155.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002094977	A1	20020718	US 2001-7407	20011204
US 2002013334	A1	20020131	US 2001-875155	20010606

PRIORITY APPLN. INFO.: US 2000-211595P P 20000615
US 2001-875155 A2 20010606
OTHER SOURCE(S): MARPAT 137:109267
GI

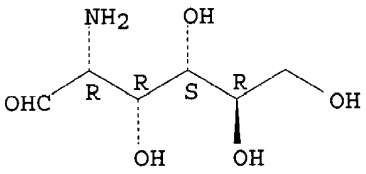
* STRUCTURE DIAGRAM TOO LARGE FOR DISPLAY - AVAILABLE VIA OFFLINE PRINT *

AB Title compds. I [X = O, S, SO, SO2, NR7; Z = HOCHCH2CH(OH)CH2CO2R3, 4-hydroxy-2-oxopyran-6-yl, etc.; n = 0, 1; R1, R2 = alkyl, arylalkyl, cycloalkyl, alkenyl, cycloalkenyl, aryl, heteroaryl, cycloheteroalkyl; R3 = H, alkyl, metal ion; R4 = H, halo, CF3, etc.; R7 = H, alkyl, aryl, alkanoyl, aroyl, alkoxycarbonyl, etc.; R9, R10 = H, alkyl], were prepd. as HMG CoA reductase inhibitors active in inhibiting cholesterol biosynthesis, modulating blood serum lipids such as lowering LDL cholesterol and/or increasing HDl cholesterol, and treating hyperlipidemia, hypercholesterolemia, hypertriglyceridemia and atherosclerosis (no data). E.g., a multistep synthesis of II is reported.

IT **3416-24-8**, Glucosamine
RL: PAC (Pharmacological activity); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(coadministered agents; prepn. of benzoxepinopyridines as HMG-CoA reductase inhibitors for the treatment of hyperlipidemia, hypercholesterolemia, hypertriglyceridemia, atherosclerosis, and other disorders)

RN 3416-24-8 HCAPLUS
CN D-Glucose, 2-amino-2-deoxy- (8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



L72 ANSWER 3 OF 19 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:858010 HCAPLUS
DOCUMENT NUMBER: 137:57085
TITLE: Effects of sugars, their methylated derivatives, and disaccharides on tumor cell migration and aggregation in vitro
AUTHOR(S): Yakshibaeva, Y. R.; Vinnitsky, V. B.

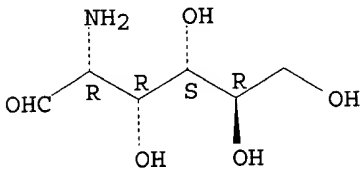
CORPORATE SOURCE: R. E. Kavetsky Inst. Experimental Pathology, Oncology Radiobiology, National Acad. Sci. Ukraine, Kiev, 03022, Ukraine
SOURCE: Eksperimental'naya Onkologiya (2001), 23(3), 161-165
CODEN: EKSODD; ISSN: 0204-3564
PUBLISHER: Institut Eksperimental'noi Patologii, Onkologii i Radiobiologii im. R. E. Kavetskogo NAN Ukrainy
DOCUMENT TYPE: Journal
LANGUAGE: Russian

AB Homotypic carcinoma 3LL cell aggregation has been inhibited by carbohydrates in the presence of spleen cells from tumor bearing mice. Carbohydrates have stimulated heterotypic aggregation of carcinoma and spleen cells. Carbohydrates inhibit migration of 3LL carcinoma and B16 melanoma cells. Spleen cells modify the carbohydrate effect on tumor cell migration in vitro.

IT **3416-24-8**, Glucosamine **7535-00-4**, Galactosamine
14307-02-9, Mannosamine
RL: PAC (Pharmacological activity); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(effects of sugars, their methylated derivs., and disaccharides on tumor cell migration and aggregation in vitro)

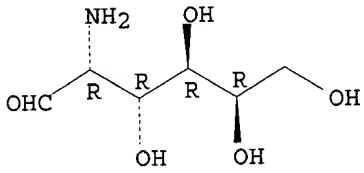
RN 3416-24-8 HCAPLUS
CN D-Glucose, 2-amino-2-deoxy- (8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



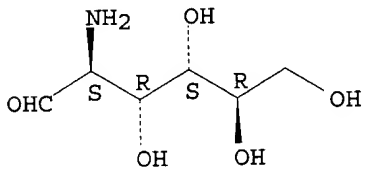
RN 7535-00-4 HCAPLUS
CN D-Galactose, 2-amino-2-deoxy- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



RN 14307-02-9 HCAPLUS
CN D-Mannose, 2-amino-2-deoxy- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L72 ANSWER 4 OF 19 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:798090 HCAPLUS
DOCUMENT NUMBER: 135:341174
TITLE: Detection and treatment of atherosclerosis based on plasma sphingomyelin concentration
INVENTOR(S): Tall, Alan R.; Jiang, Xian-Cheng
PATENT ASSIGNEE(S): Trustees of Columbia University in the City of New York, USA
SOURCE: PCT Int. Appl., 76 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001080903	A1	20011101	WO 2001-US12706	20010419

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

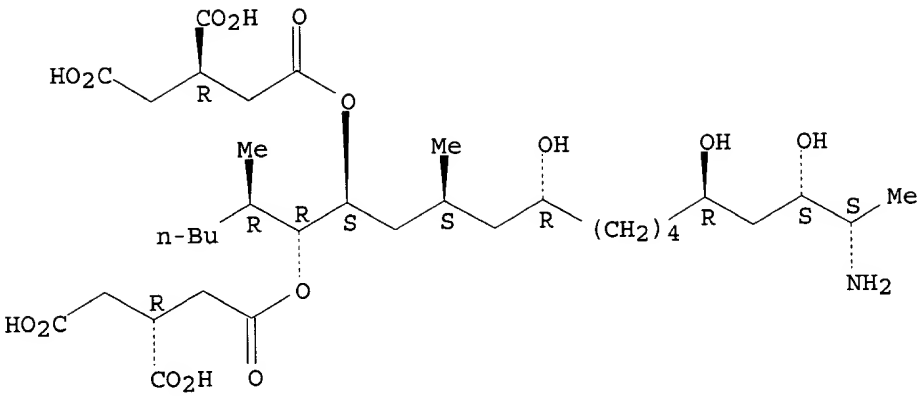
PRIORITY APPLN. INFO.: US 2000-551947 A 20000419
OTHER SOURCE(S): MARPAT 135:341174

AB The invention concerns new enzymic methods of plasma and tissue sphingomyelin concn. measurement. Also disclosed is that human plasma sphingomyelin levels are strongly pos. correlated with atherosclerosis and coronary heart disease. Thus, the use of a quick and effective plasma sphingomyelin measurement such as the subject invention, is valuable for screening assays in vitro, in cell culture or in animals to develop drugs or other treatments aimed to lower plasma sphingomyelin levels. The findings indicate that therapies aimed at reducing plasma or tissue SM levels are likely to have therapeutic benefit. These would include inhibition of sphingomyelin synthesis in the liver or arterial wall, as well as methods to enhance clearance of sphingomyelin from plasma. Thus, compds. which inhibit sphingomyelin biosynthesis or induce sphingomyelin clearance are also disclosed.

IT **116355-83-0**, **Fumonisin** B1 **121025-46-5**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(detection and treatment of atherosclerosis based on plasma sphingomyelin concn.)

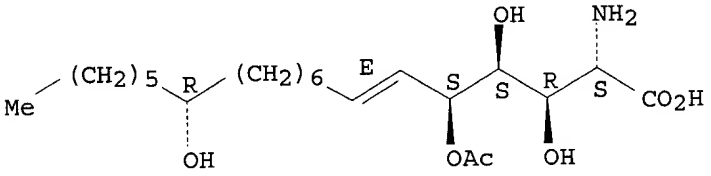
RN 116355-83-0 HCAPLUS
CN 1,2,3-Propanetricarboxylic acid, 1,1'-[(1S,2R)-1-[(2S,4R,9R,11S,12S)-12-amino-4,9,11-trihydroxy-2-methyltridecyl]-2-[(1R)-1-methylpentyl]-1,2-ethanediyl] ester, (2R,2'R)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



RN 121025-46-5 HCAPLUS
CN 6-Eicosenoic acid, 5-(acetyloxy)-2-amino-3,4,14-trihydroxy-, (2S,3R,4S,5S,6E,14R)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.



L72 ANSWER 5 OF 19 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:101286 HCAPLUS
DOCUMENT NUMBER: 134:161879
TITLE: Novel strategy for carbohydrate-based therapeutic vaccines
INVENTOR(S): Jennings, Harold J.; Sad, Subash; Guo, Zhongnu; Liu, Tianmin; Yang, Qinling
PATENT ASSIGNEE(S): National Research Council of Canada, Can.
SOURCE: PCT Int. Appl., 25 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001009298	A2	20010208	WO 2000-CA886	20000728

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

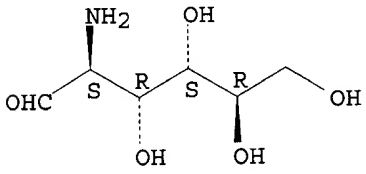
EP 1198244 A2 20020424 EP 2000-951149 20000728
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL
PRIORITY APPLN. INFO.: CA 1999-2279134 A 19990729
WO 2000-CA886 W 20000728

AB The sialic acid component of a sialic acid unit-contg. cell surface marker characteristic of cancerous mammalian cells, such as <a2-8 polysialic acid, is modified, so that cells normally expressing such a marker express instead a modified sialic acid unit-contg. cell surface marker which is strongly immunogenic. For example, the present invention enables, in a portion of patient cells which regularly express .alpha.2-8 polysialic acid (i.e. various types of cancer cells), the expression of a highly immunogenic surface antigen namely, modified .alpha.2-8 polysialic acid. The modification is suitably N-acylation of a precursor of the sialic acid, so that the N-acylated precursor becomes chem. incorporated in the polysialic acid during its intracellular biochem. synthesis. Antibodies specific for the modified antigen, which can be induced using a conjugate of a suitable portion of the modified sialic acid unit-contg. marker (such as .alpha.2-8 polysialic acid) and a protein, can then be used to eliminate cells which express <a2-8 polysialic acid. Vaccines can be prepd. utilizing conjugates of the modified sialic acid-contg. marker, or utilizing antibodies produced in response to exposure of a suitable subject to the modified sialic acid-contg. marker, for managing cancer conditions which involve cancer cells characterized, at least in part, by expression of modified sialic acid unit contg. marker.

IT **14307-02-9D**, D-Mannosamine, N-acylated
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(conjugates of modified .alpha.2-8 polysialic acid and surface antigen as therapeutic vaccines)

RN 14307-02-9 HCAPLUS
CN D-Mannose, 2-amino-2-deoxy- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L72 ANSWER 6 OF 19 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2000:227456 HCAPLUS
DOCUMENT NUMBER: 132:264090
TITLE: Therapeutic application of peptides derived from glycoprotein 10B
INVENTOR(S): Bogoch, Samuel; Bogoch, Elenore S.
PATENT ASSIGNEE(S): USA
SOURCE: PCT Int. Appl., 59 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

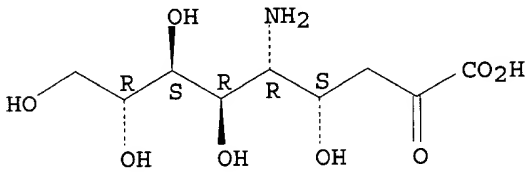
PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000018351	A2	20000406	WO 1999-US19836	19990830

WO 2000018351 A3 20000713
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
US 6242578 B1 20010605 US 1998-146755 19980904
CA 2341763 AA 20000406 CA 1999-2341763 19990830
EP 1115418 A2 20010718 EP 1999-944002 19990830
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO
PRIORITY APPLN. INFO.: US 1998-146755 A 19980904
US 1994-198139 B2 19940217
WO 1999-US19836 W 19990830

AB The authors disclose peptides derived from brain tumor-assocd. glycoprotein 10B. In one example, anti-malignin antibodies, previously described as markers of cancer transformation, are shown to increase on vaccination. Glycoconjugates of these peptides may be useful in prevention of influenza virus binding to cells, treatment of schizophrenia and diagnosing chronic viral disease assocd. with development of cancer.

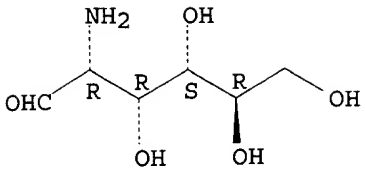
IT **114-04-5D**, Neuraminic acid, glycoprotein 10B peptide conjugates
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(for therapy)
RN 114-04-5 HCAPLUS
CN Neuraminic acid (9CI) (CA INDEX NAME)

Absolute stereochemistry.



IT **66-84-2**, D-Glucosamine hydrochloride
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(for therapy of schizophrenia in relation to dysfunctional expression of brain glycoconjugates)
RN 66-84-2 HCAPLUS
CN D-Glucose, 2-amino-2-deoxy-, hydrochloride (8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



● HCl

L72 ANSWER 7 OF 19 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1999:350607 HCAPLUS
DOCUMENT NUMBER: 131:14825
TITLE: A method of increasing nucleic acid synthesis with ultrasound
INVENTOR(S): Unger, Evan C.; McCreery, Thomas; Sadewasser, David
PATENT ASSIGNEE(S): ImaRx Pharmaceutical Corp., USA
SOURCE: PCT Int. Appl., 124 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9925385	A1	19990527	WO 1998-US23843	19981111
W: AU, CA, JP				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9913906	A1	19990607	AU 1999-13906	19981111
PRIORITY APPLN. INFO.:			US 1997-971540	19971117
			WO 1998-US23843	19981111

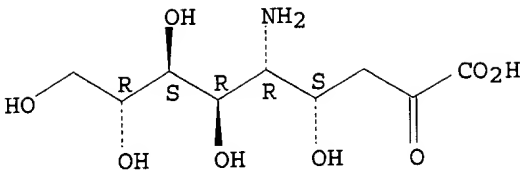
OTHER SOURCE(S): MARPAT 131:14825

AB The present invention is directed to a method of increasing nucleic acid synthesis in a cell comprising administering to the cell a therapeutically effective amt. of ultrasound for a therapeutically effective time such that said administration of said ultrasound results in said increased nucleic acid synthesis. The nucleic acid sequence may comprise an endogenous sequence or an exogenous sequence. In particular, the invention is directed to increasing the expression of stress proteins and repair proteins.

IT **114-04-5D**, Neuraminic acid, polymers contg. **3416-24-8D**, Glucosamine, polymers contg. **7535-00-4D**, Galactosamine, polymers contg.
RL: BPR (Biological process); BSU (Biological study, unclassified); BUU (Biological use, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); PROC (Process); USES (Uses)
(carrier; method of increasing nucleic acid synthesis with ultrasound)

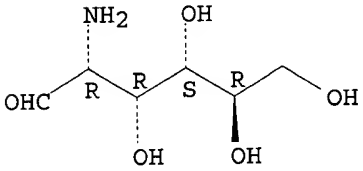
RN 114-04-5 HCAPLUS
CN Neuraminic acid (9CI) (CA INDEX NAME)

Absolute stereochemistry.



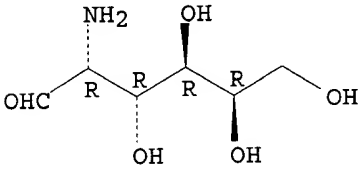
RN 3416-24-8 HCAPLUS
CN D-Glucose, 2-amino-2-deoxy- (8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



RN 7535-00-4 HCAPLUS
CN D-Galactose, 2-amino-2-deoxy- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



REFERENCE COUNT: 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L72 ANSWER 8 OF 19 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1998:604897 HCAPLUS
DOCUMENT NUMBER: 129:224944
TITLE: Preparation of acid amides and metalization of compounds
INVENTOR(S): Sinn, Hannsjorg; Maier-Borst, Wolfgang; Schrenk, Hans-hermann; Stehle, Gerd
PATENT ASSIGNEE(S): Deutsches Krebsforschungszentrum Stiftung des Offentlichen Rechts, Germany
SOURCE: PCT Int. Appl., 25 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9837057	A1	19980827	WO 1998-DE496	19980218
W: JP, US				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
DE 19706490	C1	19980917	DE 1997-19706490	19970219

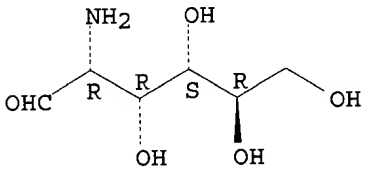
EP 966427	A1	19991229	EP 1998-912276	19980218
EP 966427	B1	20010502		
R: AT, BE, CH, DE, DK, ES, FR, GB, IT, LI, NL, SE				
AT 200896	E	20010515	AT 1998-912276	19980218
JP 2001513758	T2	20010904	JP 1998-536155	19980218
ES 2160409	T3	20011101	ES 1998-912276	19980218
US 6177561	B1	20010123	US 1999-367768	19991206
PRIORITY APPLN. INFO.:			DE 1997-19706490 A	19970219
			WO 1998-DE496 W	19980218

AB A process is disclosed for prepg. acid amides by reacting an acid with an aliph. amine in molten urea. Also disclosed is a process for metalizing compds. which can be bonded to a metal ion by reacting the compd. with a metal ion in molten urea. Also disclosed are the thus obtained products and their use for the therapy and/or diagnosis of tumors or inflammatory diseases. Thus Gd-diethylenetriaminepentaacetic acid complex was reacted with amino-.gamma.-methoxypolyethyleneglycol in molten urea in a 1:2 ratio to give the Gd complex with diamide of diethylenetriaminepentaacetic acid or tetrakis(4-sulfophenyl)porphyrin was reacted with amino-.gamma.-methoxypolyethyleneglycol in molten urea in a 1:4 ratio to give the tetraamide.

IT **3416-24-8DP**, Glucosamine, acid amide derivs., metal complexes
RL: SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(prepn. and use for therapy and/or diagnosis of tumors or inflammatory diseases)

RN 3416-24-8 HCAPLUS
CN D-Glucose, 2-amino-2-deoxy- (8CI, 9CI) (CA INDEX NAME)

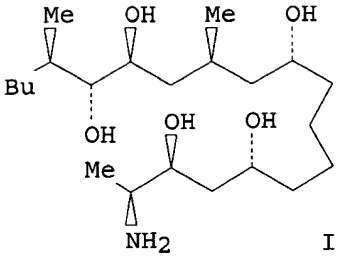
Absolute stereochemistry. Rotation (+).



REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L72 ANSWER 9 OF 19 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER:	1998:332518 HCAPLUS
DOCUMENT NUMBER:	129:81604
TITLE:	Chiron approach towards a potent toxin **fumonisin** B1 backbone: synthesis of its hexaacetate derivative
AUTHOR(S):	Gurjar, Mukund K.; Rajendran, V.; Venkatewara Rao, B.
CORPORATE SOURCE:	Inst. Inst. Chem. Technol., Hyderabad, 500 007, India
SOURCE:	Tetrahedron Letters (1998), 39(22), 3803-3806 CODEN: TELEAY; ISSN: 0040-4039
PUBLISHER:	Elsevier Science Ltd.
DOCUMENT TYPE:	Journal
LANGUAGE:	English
OTHER SOURCE(S):	CASREACT 129:81604
GI	



I

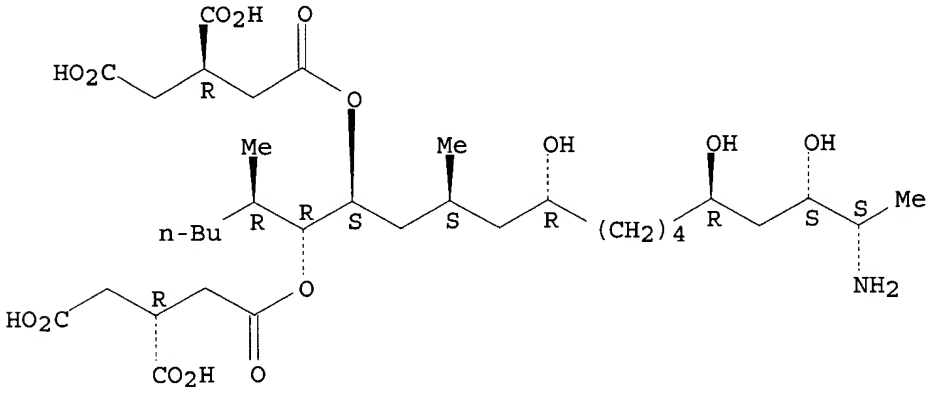
AB Synthesis of the hexaacetate of the potent toxin **fumonisin** B1-AP (I) has been described starting from carbohydrates.

IT **116355-83-0P**, **Fumonisin** B1
RL: PNU (Preparation, unclassified); PREP (Preparation)
(chiron approach to the synthesis of **fumonisin** B1-AP hexaacetate)

RN 116355-83-0 HCAPLUS

CN 1,2,3-Propanetricarboxylic acid, 1,1'-[(1S,2R)-1-[(2S,4R,9R,11S,12S)-12-amino-4,9,11-trihydroxy-2-methyltridecyl]-2-[(1R)-1-methylpentyl]-1,2-ethanediyl] ester, (2R,2'R)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

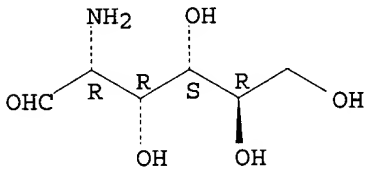


IT **3416-24-8**, D-Glucosamine
RL: RCT (Reactant); RACT (Reactant or reagent)
(chiron approach to the synthesis of **fumonisin** B1-AP hexaacetate)

RN 3416-24-8 HCAPLUS

CN D-Glucose, 2-amino-2-deoxy- (8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L72 ANSWER 10 OF 19 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1997:411073 HCAPLUS
DOCUMENT NUMBER: 127:29082
TITLE: Therapeutic method using ascorbate i.v. infusion for the treatment of cancer
INVENTOR(S): Riordan, Neil H.; Riordan, Hugh D.
PATENT ASSIGNEE(S): Center for the Improvement of Human Functioning Int'l, Inc., USA
SOURCE: U.S., 6 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5639787	A	19970617	US 1995-397663	19950228

PRIORITY APPLN. INFO.: US 1995-397663 19950228

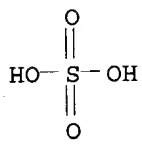
AB A method of treating cancer in a patient is provided which includes raising and maintaining the concn. of ascorbic acid, or ascorbate, in the patient's plasma to at least the level expected to be toxic to an in vitro culture of cells of the type of cancer being treated, the required plasma ascorbate levels being achieved and maintained using long term i.v. infusions of large amts. of ascorbate, with or without ascorbate cytotoxicity effectiveness enhancing or tumor site delivery and absorption enhancing agents.

IT **29031-19-4**, Glucosamine sulfate
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(ascorbate i.v. infusion, alone or with other agents, for cancer treatment)

RN 29031-19-4 HCAPLUS
CN D-Glucose, 2-amino-2-deoxy-, sulfate (salt) (8CI, 9CI) (CA INDEX NAME)

CM 1

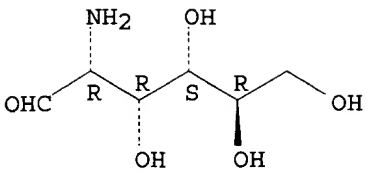
CRN 7664-93-9
CMF H2 O4 S



CM 2

CRN 3416-24-8
CMF C6 H13 N O5

Absolute stereochemistry. Rotation (+).



L72 ANSWER 11 OF 19 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1997:311165 HCAPLUS
DOCUMENT NUMBER: 126:327558
TITLE: Radiation sensitization using texaphyrins for treatment of neoplasms or atheromas
INVENTOR(S): Sessler, Jonathan L.; Harriman, Anthony M.; Miller, Richard A.
PATENT ASSIGNEE(S): Pharmacyclics, Inc., USA; Board of Regents, Univ. of Tex. Sys.
SOURCE: U.S., 39 pp., Cont.-in-part of U.S. 5,457,183.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 21
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5622946	A	19970422	US 1995-437968	19950510
US 5457183	A	19951010	US 1993-135118	19931012
US 5583220	A	19961210	US 1995-449681	19950524
US 5580543	A	19961203	US 1995-458267	19950602
US 5587371	A	19961224	US 1995-458909	19950602
US 5632970	A	19970527	US 1995-486967	19950607
US 5801229	A	19980901	US 1996-713701	19960913
US 5888997	A	19990330	US 1997-795393	19970204
US 5969111	A	19991019	US 1997-775261	19970204
US 6069140	A	20000530	US 1997-970864	19971114
US 6072038	A	20000606	US 1998-104870	19980625

PRIORITY APPLN. INFO.:

US 1993-135118	A2	19931012
US 1989-320293	A3	19890306
US 1990-539975	A2	19900618
US 1991-771393	B2	19910930
US 1992-822064	A2	19920121
US 1992-822964	A2	19920121
US 1993-75123	B2	19930609
US 1993-98514	A1	19930728
US 1994-227370	A2	19940414
US 1995-227370	A2	19940414
WO 1994-US6284	A1	19940609
WO 1994-US11491	A1	19941012
US 1995-437968	A3	19950510
US 1995-452261	B2	19950526
US 1996-679162	A2	19960710
US 1996-713701	A1	19960913
US 1997-795393	A1	19970204

OTHER SOURCE(S): MARPAT 126:327558
AB Texaphyrins are provided for use as radiation sensitizers. Advantageous properties of texaphyrins for use as a radiation sensitizer include: (1) a low redox potential, which allows radiation-induced hydrated electrons to

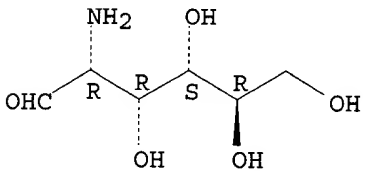
flow to texaphyrin rather than neutralizing hydroxyl radicals, allowing hydroxyl radicals to cause cellular damage; (2) a relatively stable texaphyrin radical that reacts readily to covalently modify neighboring mols., causing further cellular damage; (3) intrinsic biolocalization; and (4) indifference to the presence or absence of O2. These properties allow texaphyrins to be particularly effective for treating the hypoxic areas of solid neoplasms. Methods of treatment for an individual having a neoplasm or atheroma include the use of a texaphyrin as a radiation sensitizer and as an agent for photodynamic tumor therapy, or the use of a texaphyrin for internal and for external ionizing radiation. Novel texaphyrins are provided.

IT **3416-24-8D**, Glucosamine, texaphyrin conjugates, metal complexes
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(texaphyrins for radiation sensitizers and photodynamic therapy)

RN 3416-24-8 HCAPLUS

CN D-Glucose, 2-amino-2-deoxy- (8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



L72 ANSWER 12 OF 19 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1996:718329 HCAPLUS

DOCUMENT NUMBER: 126:1216

TITLE: Abnormal glycoconjugates as diagnostics of disease processes and their use as decoy substances in therapy

INVENTOR(S): Bogoch, Samuel; Bogoch, Elenore S.

PATENT ASSIGNEE(S): USA

SOURCE: PCT Int. Appl., 36 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9632106	A1	19961017	WO 1995-US4553	19950411
W: AU, CA, JP				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9522891	A1	19961030	AU 1995-22891	19950411
EP 812191	A1	19971217	EP 1995-916363	19950411
R: CH, DE, FR, GB, LI				

PRIORITY APPLN. INFO.: WO 1995-US4553 19950411

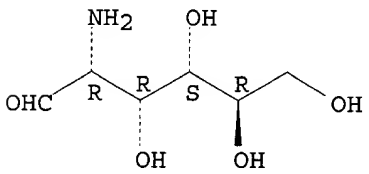
AB This invention concerns products and methods for the diagnosis and treatment of disorders of conjugated carbohydrate constituents which contribute to cell dysfunction and cell death. The invention teaches that where normal carbohydrate constituents are covalently bound with other cell structures in the form of glycoconjugates, these carbohydrate constituents contribute to cell stability, to receptor and recognition functions of the cell, and to the protection of cell constituents from

damage. When these carbohydrates are reduced in concn. or structurally altered, together defined as aglyco states, the stability, receptor, recognition, and protective functions of these carbohydrates are diminished or lost, and cell dysfunction and death result, with disease states. These disease states, in the nervous system for example, include dementias (as in schizophrenia and brain tumors), Parkinsonism and Alzheimer's Disease. These disorders can be diagnosed 1) by direct detn. of structural changes in the nervous system glycoconjugates; or 2) because these aglyco products may act as antigens, by the detn. of antibodies produced by the body against the aglyco products. Antibodies produced by the body against aglyco products can have a deleterious effect (e.g. in normal developing brain) or desirable effect (e.g. in brain tumors). The invention includes glyco decoys, which are glycoconjugate substances which act as artificial receptors for viruses and other pathogens which would normally attach to and enter the body's cells, and methods for the prodn. and detection of aglyco products which are glycoconjugate products with reduced or altered carbohydrate constituents and the aglyco antibodies produced against these aglyco products. The invention also includes a treatment for cancer consisting of the use of antimalignin antibodies or derivs. thereof which can kill or inhibit the growth of cancer cells.

IT **66-84-2**, D-Glucosamine hydrochloride
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(abnormal glycoconjugates as diagnostics of disease processes and their use as decoy substances in therapy)

RN 66-84-2 HCAPLUS
CN D-Glucose, 2-amino-2-deoxy-, hydrochloride (8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).

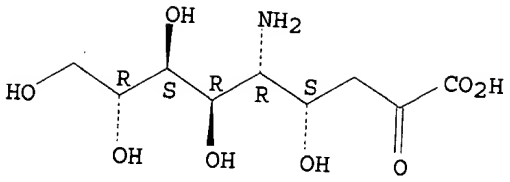


● HCl

IT **114-04-5**, Neuraminic acid
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); OCCU (Occurrence); USES (Uses)
(glycoconjugates contg.; abnormal glycoconjugates as diagnostics of disease processes and their use as decoy substances in therapy)

RN 114-04-5 HCAPLUS
CN Neuraminic acid (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L72 ANSWER 13 OF 19 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1996:694251 HCAPLUS
DOCUMENT NUMBER: 125:326402
TITLE: An immunoreactive conjugate, method for its preparation, antibodies to the conjugate and a pharmaceutical composition and diagnostic device containing them
INVENTOR(S): Maes, Roland
PATENT ASSIGNEE(S): Anda Biologicals S.A., Fr.
SOURCE: Eur. Pat. Appl., 19 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: French
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 736770	A2	19961009	EP 1996-870042	19960401
EP 736770	A3	19970502		
R: BE, DE, FR, GB, IT				
BE 1009230	A6	19970107	BE 1995-316	19950405
BE 1009917	A6	19971104	BE 1996-113	19960208

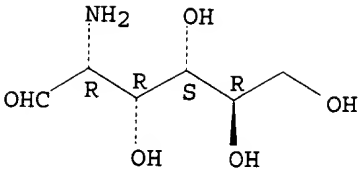
PRIORITY APPLN. INFO.: BE 1995-316 19950405
BE 1996-113 19960208

AB An immunoreactive conjugate is disclosed which contains 1 or more haptens consisting of a sulfhydryl group and one of the following: amino acids, carbohydrates, amino carbohydrates, phosphatidylinositol, sphingosine, and their nitrosyl, acyl, or acetyl derivs., the haptens being coupled to a protein with a mol. wt. >8000 Kd and/or a solid support by a coupling agent capable of binding to the sulfhydryl group of the hapten. Thus, NO-cysteine and NO-N-acetyl-L-cysteine conjugates with albumin were prepd., and birds and mammals were vaccinated. IgG and IgM class antibodies specific for N-acetyl-L-cysteine were detected in the subjects. Addnl. analyses demonstrated that many HIV-pos. patients have IgG specific for acetyl-cysteine. Pharmaceutical compns. using these immunoreactive conjugates can be used in the prevention and/or treatment of autoimmunity, AIDS, cancer, tuberculosis and a variety of other diseases.

IT **3416-24-8**, Glucosamine **7535-00-4**, Galactosamine
14307-02-9, Mannosamine
RL: BPR (Biological process); BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); PROC (Process); USES (Uses)
(in prepn. of immunoreactive conjugates with haptens and carrier protein, antibodies to them, and application in diagnosis and treatment of disease)

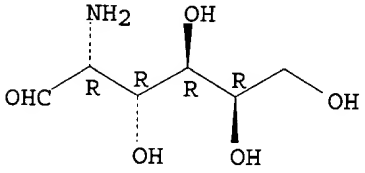
RN 3416-24-8 HCAPLUS
CN D-Glucose, 2-amino-2-deoxy- (8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



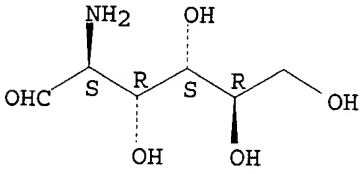
RN 7535-00-4 HCAPLUS
CN D-Galactose, 2-amino-2-deoxy- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



RN 14307-02-9 HCAPLUS
CN D-Mannose, 2-amino-2-deoxy- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



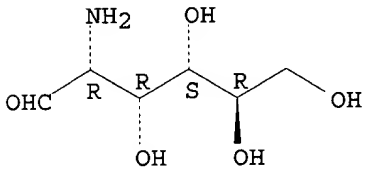
L72 ANSWER 14 OF 19 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1996:170076 HCAPLUS
DOCUMENT NUMBER: 124:256512
TITLE: Pathways of glycosphingolipid biosynthesis in SW13 cells in the presence and absence of vimentin intermediate filaments
AUTHOR(S): Gillard, Baiba K.; Harrell, Rhonda G.; Marcus, Donald M.
CORPORATE SOURCE: Dep. Med., Baylor Coll. Med., Houston, TX, 77030, USA
SOURCE: Glycobiology (1996), 6(1), 33-42
CODEN: GLYCE3; ISSN: 0959-6658
PUBLISHER: Oxford University Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Incorporation of sugars into glycosphingolipids (GSL) is diminished in SW13 cells that lack a vimentin intermediate filament (IF) network (vim-) compared to vim+ cells. To further analyze the nature of this abnormality, cells were double-labeled with 3H-serine and 14C-sugars. There was no difference between vim+ and vim- cells in the incorporation of serine into GSI, although the usual difference in sugar incorporation was obsd. This indicated that the defect in vim- cells was not in the incorporation of sugars into ceramide synthesized de novo by acylation of sphinganine (pathway 1). Sugars can also be incorporated into ceramide synthesized from sphingosine that is derived from catabolism of

sphingolipids (pathway 2), and into GSL that recycle through the Golgi app. from endosomes (pathway 3). The amt. of galactose and glucosamine incorporated into GSL in these 3 pathways was analyzed by the use of 2 inhibitors of sphingolipid biosynthesis. .beta.-Chloroalanine inhibits the de novo synthesis of sphinganine (pathway 1), and **fumonisin** B1 inhibits the acylation of sphinganine and sphingosine (pathways 1 and 2). In both vin+ and vin- cells, only 26-40% of sugar incorporation into GSL took place in pathway 1, and 60-80% of sugar incorporation took place in the recycling pathways. Moreover, in contrast to larger GSL, GlcCer was not synthesized in pathway 3. These observations indicate that vimentin IF facilitate the recycling of GSL and sphingosine, and that the differences between vim+ and vim- cells are predominantly in pathway 2 and 3. Furthermore, although it is generally believed that virtually all GSL are synthesized in the de novo pathway, these data indicate that the recycling pathways predominate in the incorporation of sugars into GSL in SW13 cells.

IT **3416-24-8**, Glucosamine
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(pathways of glycosphingolipid biosynthesis in SW13 cells in the presence and absence of vimentin intermediate filaments)

RN 3416-24-8 HCAPLUS
CN D-Glucose, 2-amino-2-deoxy- (8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



L72 ANSWER 15 OF 19 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1993:225689 HCAPLUS
DOCUMENT NUMBER: 118:225689
TITLE: Method of altering sphingolipid metabolism and detecting **fumonisin** ingestion and contamination
INVENTOR(S): Merrill, Alfred H., Jr.; Wang, Elaine W.; Liotta, Dennis C.; Riley, Ronald T.
PATENT ASSIGNEE(S): Emory University, USA; United States Dept. of Agriculture
SOURCE: PCT Int. Appl., 72 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9302673	A1	19930218	WO 1992-US6460	19920805
W: CA, JP				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, SE				
US 5232837	A	19930803	US 1991-740426	19910805
US 5518879	A	19960521	US 1993-42147	19930402
US 6127578	A	20001003	US 1996-627499	19960404

PRIORITY APPLN. INFO.: US 1991-740426 A 19910805
US 1993-42147 A3 19930402

OTHER SOURCE(S): MARPAT 118:225689

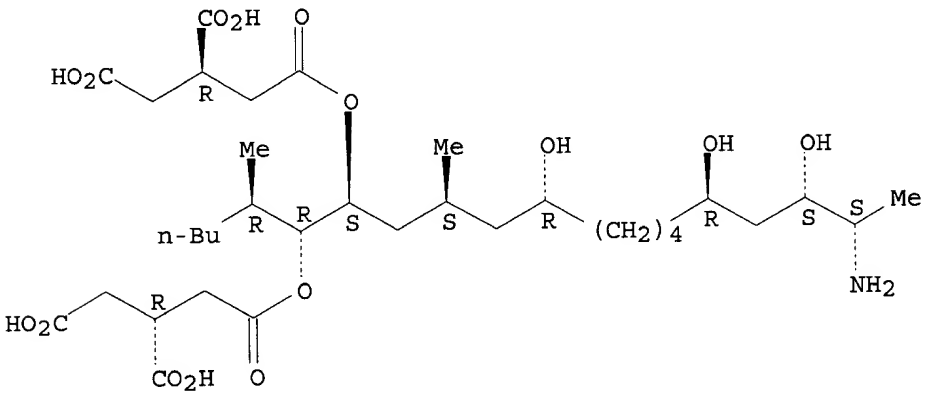
AB **Fumonisins**, mycotoxins from Fusarium moniliforme structurally resembling sphingosine, disrupt sphingosine metab. and inhibit de novo sphingolipid biosynthesis in animals; these compds. and synthetic analogs are therefore useful in treatment of disorders in sphingolipid metab. One site of action is ceramide synthetases, to which **fumonisins** bind, thereby inhibiting conversion of sphinganine to dihydroceramide or of sphingosine to ceramide. Ingestion of **fumonisins**, e.g. by livestock with F. moniliforme-infected grain, and disorders in sphingolipid metab. are detected by detn. of appropriate metabolic indicators such as sphinganine and ceramide levels. Thus, hepatotoxic and hepatocarcinogenic **fumonisin** B1 almost completely inhibited incorporation of label from serine-14C into sphingosine by rat hepatocytes in vitro without affecting formation of other phospholipids. L-Alanine Me ester was converted in 7 steps, via Grignard reaction of N-protected 4-amino-2-pentenal with C13H27MgBr, to 2-aminooctadeca-3,5-diol, a **fumonisin** analog.

IT **116355-83-0**, **Fumonisin** B1
RL: BIOL (Biological study)
(sphingolipid metab. inhibition by)

RN 116355-83-0 HCAPLUS

CN 1,2,3-Propanetricarboxylic acid, 1,1'-[(1S,2R)-1-[(2S,4R,9R,11S,12S)-12-amino-4,9,11-trihydroxy-2-methyltridecyl]-2-[(1R)-1-methylpentyl]-1,2-ethanediyl] ester, (2R,2'R)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



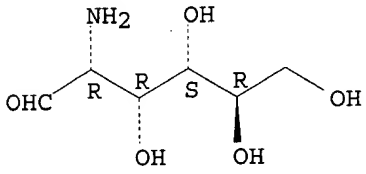
L72 ANSWER 16 OF 19 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1975:132803 HCAPLUS
DOCUMENT NUMBER: 82:132803
TITLE: In vitro cytotoxic effect of some saccharide phenylhydrazone derivatives
AUTHOR(S): Fuska, J.; Linek, K.; Buzinkay, S.
CORPORATE SOURCE: Chem. Technol. Fac., Slovak Technol. Univ., Bratislava, Czech.
SOURCE: Neoplasma (1974), 21(5), 561-8
CODEN: NEOLA4; ISSN: 0028-2685
DOCUMENT TYPE: Journal
LANGUAGE: English
GI For diagram(s), see printed CA Issue.
AB Of 40 compds. tested for their effects on precursor incorporation into

proteins and nucleic acids of cellular fractions of Ehrlich ascites carcinoma (EAC), tri-O-acetyl-D-erythrose p-nitrophenylhydrazone (I) [54420-05-2] was the most effective, depressing the incorporation of adenine, thymidine, uridine, and valine into the EAC cells at concns. <100 .mu.g/ml. I at 3.5-10.7 .mu.g/ml also repressed the proliferation of EAC cells, L5178, and NK/Ly. Tri-O-acetyl-D-treose p-nitrophenylhydrazine [54420-06-3], tetra-O-acetyl-D-arabinose 2,4-dinitrophenylhydrazine [54420-07-4], and penta-O-acetyl-D-galactose p-nitrophenylhydrazine [14155-25-0] were also cytotoxic.

IT **66-84-2**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(neoplasm inhibitor)

RN 66-84-2 HCAPLUS
CN D-Glucose, 2-amino-2-deoxy-, hydrochloride (8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



● HCl

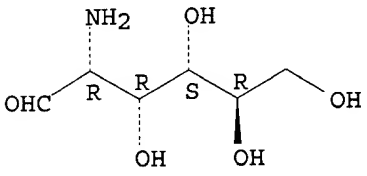
L72 ANSWER 17 OF 19 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1972:428993 HCAPLUS
DOCUMENT NUMBER: 77:28993
TITLE: Inhibition of mouse ascites tumors by carbohydrate combined with immunization
AUTHOR(S): Eng, C. P.; Bhatnagar, M. K.; Morgan, J. F.
CORPORATE SOURCE: Dep. Cancer Res., Univ. Saskatchewan, Saskatoon, SK, Can.
SOURCE: Canadian Journal of Physiology and Pharmacology (1972), 50(2), 156-63
CODEN: CJPPA3; ISSN: 0008-4212
DOCUMENT TYPE: Journal
LANGUAGE: English

AB In mice inoculated with TA3 ascites tumor cells daily i.p. injection with D-mannose [3458-28-4] had no effect, whereas D-glucosamine-HCl [66-84-2] decreased the total tumor vol. but not the packed cell vol.; 2-deoxy-D-glucose [154-17-6] moderately decreased the tumor fluid and packed cell vol. and DL-glyceraldehyde (I) [56-82-6] drastically reduced tumor development. With multiple injections on a single day, 2-deoxy-D-glucose produced no effect, D-glucosamine caused a moderate inhibition, and I completely inhibited tumor development. Previous immunization of mice with an insol. fraction prepd. from the tumor cells, potentiated the inhibitory effects of I. This latter effect was also true with Ehrlich, Ehrlich-Lettre, 6C3HED, and SAI mouse ascites tumors. The LD50 of I in mice was 3.0 g/kg. Mice injected i.p. with I (1% soln.) developed enlarged livers which showed excessive amts. of cytoplasmic glycoprotein granules.

IT **66-84-2**

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(neoplasm inhibition by, immunization with tumor exts. in relation to)
RN 66-84-2 HCAPLUS
CN D-Glucose, 2-amino-2-deoxy-, hydrochloride (8CI, 9CI) (CA INDEX NAME)

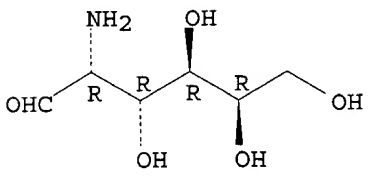
Absolute stereochemistry. Rotation (+).



● HCl

L72 ANSWER 18 OF 19 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1971:461683 HCAPLUS
DOCUMENT NUMBER: 75:61683
TITLE: Cytotoxic effects of exogenous D-galactosamine on experimental tumors
AUTHOR(S): St. Arneault, G.; Walter, L.; Bekesi, J. G.
CORPORATE SOURCE: Dep. Med. A, Roswell Park Mem. Inst., Buffalo, NY, USA
SOURCE: International Journal of Cancer (1971), 7(3), 483-90
CODEN: IJCNAW; ISSN: 0020-7136
DOCUMENT TYPE: Journal
LANGUAGE: English
AB D-Galactosamine (I) inhibited the viability and the transplantability of mouse Ehrlich carcinoma cells in vitro. The cytotoxic effect was proportional with the I conc., and was not affected by glucose or pyruvate. I was taken up rapidly by the tumor cell. Of the intracellular acid-sol. I, 98% was not phosphorylated. I inhibited DNA synthesis in Sarcoma-180 ascites tumor cells by 90%, but had min. effect on such synthesis in normal tissues.
IT **7535-00-4**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(neoplasm inhibition by)
RN 7535-00-4 HCAPLUS
CN D-Galactose, 2-amino-2-deoxy- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



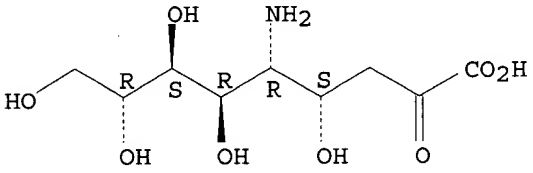
L72 ANSWER 19 OF 19 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1962:478809 HCAPLUS
DOCUMENT NUMBER: 57:78809
ORIGINAL REFERENCE NO.: 57:15699c-d
TITLE: Abnormalities 3in lipid metabolism in two members of a family with Niemann-Pick disease
AUTHOR(S): Cumings, J. N.
CORPORATE SOURCE: Inst. Neurol., London
SOURCE: Cerebral Sphingolipidoses, Symp. TaySachs' Disease Allied Disorders, New York, N. Y. (1962), 1961, 171-8
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable

AB The two children were a boy who died at 6 and his sister at 8.5 years. A marked loss in cerebral total phospholipids was shown in the boy but not his sister. Both showed an increase in sphingomyelin, esp. in the spleen where the phospholipid content was also raised. Total cholesterol levels were raised and the neuraminic acid figure of the cerebral cortex indicated a ganglioside content of nearly 2% which was above normal in each child. The neutral cerebrosides and the sulfatide content of the white matter were normal, 10.5 g./100 g. and 1.7 g./100 g. dry wt., resp.

IT **114-04-5**, Neuraminic acid
(metabolism of, in Niemann-Pick disease)
RN 114-04-5 HCAPLUS
CN Neuraminic acid (9CI) (CA INDEX NAME)

Absolute stereochemistry.



Add'l Databases Searched: Medline, Embase, Biosis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 FILE LAST UPDATED: 2 FEB 2003 (20030202/UP). FILE COVERS 1958 TO DATE.

 On June 9, 2002, MEDLINE was reloaded. See HELP RLOAD for details.

 MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the
 MeSH 2003 vocabulary. See http://www.nlm.nih.gov/mesh/summ2003.html
 for a description on changes.

 This file contains CAS Registry Numbers for easy and accurate
 substance identification.
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— Same Str. search as before

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L1                STR
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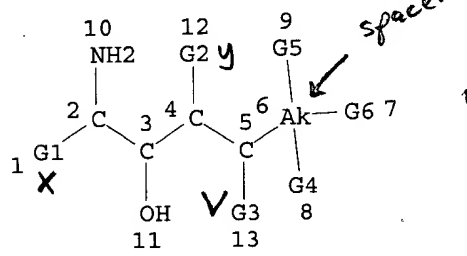

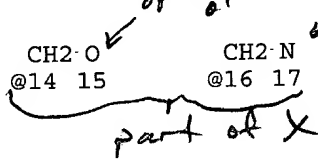


```
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V - VAR G3=H/O
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    VAR G5=H/OH
    VAR G6=H/C/N/O
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    CONNECT IS E3  RC AT   4
    CONNECT IS E3  RC AT   5
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    DEFAULT ECLEVEL IS LIMITED
    ECOUNT  IS M1-X20 C  AT   6
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— O is open allowing for hydroxyl or ester

— allows spacer to be alkyl ($G_4 = G_5 = H$) hydroxyalkyl (one of G_4 & G_5 = OH) or dihydroxyalkyl (both G_4 & G_5 = OH).

— limits generic alkyl group at 6 to having 1–20 carbons

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    NUMBER OF NODES IS  17

    STEREO ATTRIBUTES: NONE
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    L3                SCR 1146
    L4                SCR 1700
    L5                SCR 1568
    L6                SCR 2043
    L7           909 SEA FILE=REGISTRY SSS FUL L1 AND L2 AND L3 AND L4 AND L5 NOT
                     L6
    L73           30 SEA FILE=REGISTRY ABB=ON  PLU=ON  L7 AND MEDLINE/LC
    L77         9319 SEA FILE=MEDLINE ABB=ON  PLU=ON  L73
    L85        60062 SEA FILE=MEDLINE ABB=ON  PLU=ON  ANTINEOPLASTIC AGENTS/CT (L)
                     (AD OR DT OR PD OR PK OR TU)/CT
    L86        38047 SEA FILE=MEDLINE ABB=ON  PLU=ON  L85/MAJ
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— finds structures present in Medline

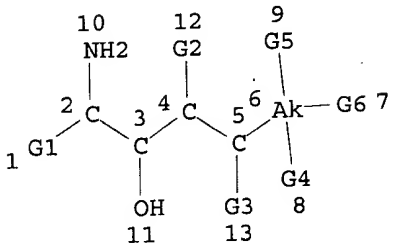
← Search structures in Medline

= indexed as Major topic of paper

Searched by Thom Larson, STIC, 308-7309

L87 18 SEA FILE=MEDLINE ABB=ON PLU=ON L77 AND L86

=> d que l93
L1 STR



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CONNECT IS X4 RC AT 6
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DEFAULT ECLEVEL IS LIMITED
ECOUNT IS M1-X20 C AT 6

GRAPH ATTRIBUTES:
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NUMBER OF NODES IS 17

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L4 SCR 1700
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L6 SCR 2043
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L77 9319 SEA FILE=MEDLINE ABB=ON PLU=ON L73
L90 6894 SEA FILE=MEDLINE ABB=ON PLU=ON SPHINGOLIPIDOSES+NT,PFT/CT
L92 5515 SEA FILE=MEDLINE ABB=ON PLU=ON L90/MAJ
L93 21 SEA FILE=MEDLINE ABB=ON PLU=ON L92 AND L77

generic to Tay-Sachs & Niemann-Picks

=> s l87 or l93
L115 39 L87 OR L93 combine answer sets

=> file embase
FILE 'EMBASE' ENTERED AT 17:08:52 ON 03 FEB 2003

FILE COVERS 1974 TO 30 Jan 2003 (20030130/ED)

EMBASE has been reloaded. Enter HELP RLOAD for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

structure search as Same ∩ before.

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L1                STR
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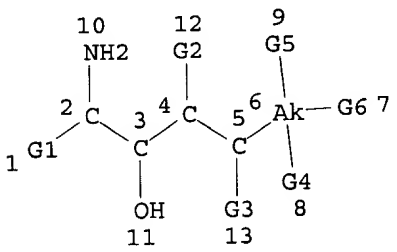

CH2·O @14 15

CH2·N @16 17

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VAR G5=H/OH
VAR G6=H/C/N/O
NODE ATTRIBUTES:
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CONNECT IS E3  RC AT   3
CONNECT IS E3  RC AT   4
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DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED
ECOUNT  IS M1-X20 C  AT   6

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS  17

STEREO ATTRIBUTES: NONE
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L4               SCR 1700
L5               SCR 1568
L6               SCR 2043
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L94       493651 SEA FILE=EMBASE ABB=ON  PLU=ON  ANTINEOPLASTIC AGENT+NT,PFT/CT

L95          3907 SEA FILE=EMBASE ABB=ON  PLU=ON  L74
L97        112753 SEA FILE=EMBASE ABB=ON  PLU=ON  L94/MAJ (L) (DT OR PC)/CT
L98            10 SEA FILE=EMBASE ABB=ON  PLU=ON  L95 AND L97
```

DT = Drug Therapy
PC = Pharmacology

```
=> d que l100
L1                STR
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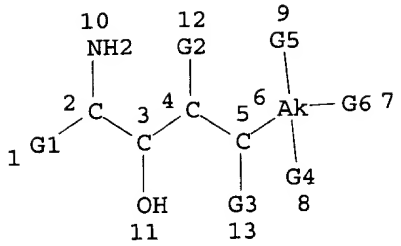

CH2 O
@14 15

CH2 N
@16 17

VAR G1=H/CH3/14/16
VAR G2=H/O
VAR G3=H/O
VAR G4=H/OH
VAR G5=H/OH
VAR G6=H/C/N/O
NODE ATTRIBUTES:
CONNECT IS E3 RC AT 2
CONNECT IS E3 RC AT 3
CONNECT IS E3 RC AT 4
CONNECT IS E3 RC AT 5
CONNECT IS X4 RC AT 6
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED
ECOUNT IS M1-X20 C AT 6

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS 17

STEREO ATTRIBUTES: NONE

```
L2                SCR 405
L3                SCR 1146
L4                SCR 1700
L5                SCR 1568
L6                SCR 2043
L7            909 SEA FILE=REGISTRY SSS FUL L1 AND L2 AND L3 AND L4 AND L5 NOT
                  L6
L74             8 SEA FILE=REGISTRY ABB=ON  PLU=ON  L7 AND EMBASE/LC
L95          3907 SEA FILE=EMBASE ABB=ON  PLU=ON  L74
L99         11814 SEA FILE=EMBASE ABB=ON  PLU=ON  LIPIDOSIS+NT,PFT/CT
L100            4 SEA FILE=EMBASE ABB=ON  PLU=ON  L95 AND L99
```

{ In EMBASE controlled terms - generic to Tay-Sachs & Niemann Pick

=> s l98 or l100
L116 14 L98 OR L100

combine EMBASE answer sets.

=> File biosis
FILE 'BIOSIS' ENTERED AT 17:10:02 ON 03 FEB 2003
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FILE COVERS 1969 TO DATE.
CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT
FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 29 January 2003 (20030129/ED)

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=> d que l103
L1                STR
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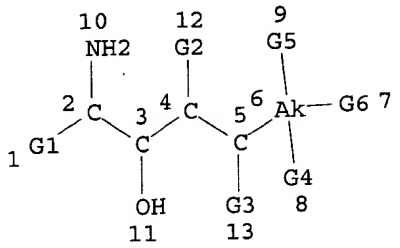
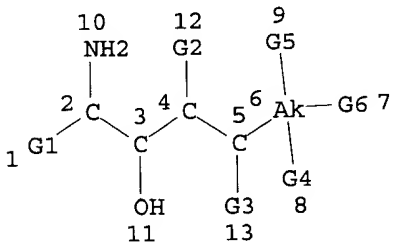



```
VAR G1=H/CH3/14/16
VAR G2=H/O
VAR G3=H/O
VAR G4=H/OH
VAR G5=H/OH
VAR G6=H/C/N/O
NODE ATTRIBUTES:
CONNECT IS E3  RC AT   2
CONNECT IS E3  RC AT   3
CONNECT IS E3  RC AT   4
CONNECT IS E3  RC AT   5
CONNECT IS X4  RC AT   6
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED
ECOUNT  IS M1-X20 C  AT   6

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS  17

STEREO ATTRIBUTES: NONE
L2               SCR 405
L3               SCR 1146
L4               SCR 1700
L5               SCR 1568
L6               SCR 2043
L7           909 SEA FILE=REGISTRY SSS FUL L1 AND L2 AND L3 AND L4 AND L5 NOT
                 L6
L75           43 SEA FILE=REGISTRY ABB=ON  PLU=ON  L7 AND BIOSIS/LC
L101        5636 SEA FILE=BIOSIS ABB=ON  PLU=ON  L75
L102        1864 SEA FILE=BIOSIS ABB=ON  PLU=ON  ANTINEOPLASTIC AGENT/CT OR
                 ANTINEOPLASTIC AGENTS/CT
L103           1 SEA FILE=BIOSIS ABB=ON  PLU=ON  L102 AND L101


=> d que l105
L1                STR
```

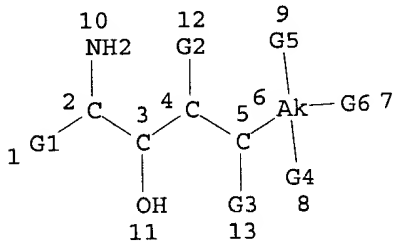


```
VAR G1=H/CH3/14/16
VAR G2=H/O
VAR G3=H/O
VAR G4=H/OH
VAR G5=H/OH
VAR G6=H/C/N/O
NODE ATTRIBUTES:
CONNECT IS E3  RC AT   2
CONNECT IS E3  RC AT   3
CONNECT IS E3  RC AT   4
CONNECT IS E3  RC AT   5
CONNECT IS X4  RC AT   6
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED
ECOUNT  IS M1-X20 C  AT   6

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS  17

STEREO ATTRIBUTES: NONE
L2                SCR 405
L3                SCR 1146
L4                SCR 1700
L5                SCR 1568
L6                SCR 2043
L7           909  SEA FILE=REGISTRY SSS FUL L1 AND L2 AND L3 AND L4 AND L5 NOT
                  L6
L75           43  SEA FILE=REGISTRY ABB=ON  PLU=ON  L7 AND BIOSIS/LC
L101        5636  SEA FILE=BIOSIS ABB=ON  PLU=ON  L75
L104         263  SEA FILE=BIOSIS ABB=ON  PLU=ON  ANTICANCER AGENT/CT OR
                  ANTICANCER AGENTS/CT
L105           0  SEA FILE=BIOSIS ABB=ON  PLU=ON  L101 AND L104


=> d que l107
L1                STR
```



CH2·O @14 15

CH2·N @16 17

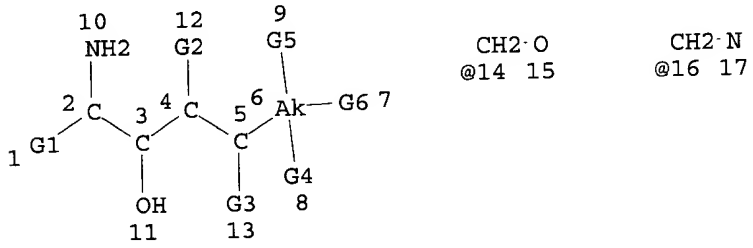
```
VAR G1=H/CH3/14/16
VAR G2=H/O
VAR G3=H/O
VAR G4=H/OH
VAR G5=H/OH
VAR G6=H/C/N/O
NODE ATTRIBUTES:
CONNECT IS E3  RC AT   2
CONNECT IS E3  RC AT   3
CONNECT IS E3  RC AT   4
CONNECT IS E3  RC AT   5
```

CONNECT IS X4 RC AT 6
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED
ECOUNT IS M1-X20 C AT 6

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS 17

STEREO ATTRIBUTES: NONE
L2 SCR 405
L3 SCR 1146
L4 SCR 1700
L5 SCR 1568
L6 SCR 2043
L7 909 SEA FILE=REGISTRY SSS FUL L1 AND L2 AND L3 AND L4 AND L5 NOT L6
L75 43 SEA FILE=REGISTRY ABB=ON PLU=ON L7 AND BIOSIS/LC
L101 5636 SEA FILE=BIOSIS ABB=ON PLU=ON L75
L106 21825 SEA FILE=BIOSIS ABB=ON PLU=ON NEOPLASM/CT OR NEOPLASMS/CT
L107 4 SEA FILE=BIOSIS ABB=ON PLU=ON L101 AND L106

=> d que l109
L1 STR



VAR G1=H/CH3/14/16
VAR G2=H/O
VAR G3=H/O
VAR G4=H/OH
VAR G5=H/OH
VAR G6=H/C/N/O
NODE ATTRIBUTES:
CONNECT IS E3 RC AT 2
CONNECT IS E3 RC AT 3
CONNECT IS E3 RC AT 4
CONNECT IS E3 RC AT 5
CONNECT IS X4 RC AT 6
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED
ECOUNT IS M1-X20 C AT 6

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS 17

STEREO ATTRIBUTES: NONE
L2 SCR 405
L3 SCR 1146
L4 SCR 1700

```
L5              SCR 1568
L6              SCR 2043
L7          909 SEA FILE=REGISTRY SSS FUL L1 AND L2 AND L3 AND L4 AND L5 NOT
                L6
L75          43 SEA FILE=REGISTRY ABB=ON  PLU=ON  L7 AND BIOSIS/LC
L101       5636 SEA FILE=BIOSIS ABB=ON  PLU=ON  L75
L108       1311 SEA FILE=BIOSIS ABB=ON  PLU=ON  LIPIPIDOS!S/CT OR SPHINGOLIPIDO
                S!S/CT OR TAY SACH?/CT OR TAY-SACH? OR NEIMANN PICK?/CT OR
                NEIMAN-PICK?/CT
L109          4 SEA FILE=BIOSIS ABB=ON  PLU=ON  L101 AND L108


=> s l103 or l105 or l107 or l109
L117          9 L103 OR L105 OR L107 OR L109

=> dup rem l115 l116 l117
FILE 'MEDLINE' ENTERED AT 17:19:30 ON 03 FEB 2003

FILE 'EMBASE' ENTERED AT 17:19:30 ON 03 FEB 2003
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FILE 'BIOSIS' ENTERED AT 17:19:30 ON 03 FEB 2003
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PROCESSING COMPLETED FOR L115
PROCESSING COMPLETED FOR L116
PROCESSING COMPLETED FOR L117
L122         59 DUP REM L115 L116 L117 (3 DUPLICATES REMOVED)

=> D IBIB ABS HITRN 1-59
'HITRN' IS NOT A VALID FORMAT
In a multifile environment, a format can only be used if it is valid
in at least one of the files.  Refer to file specific help messages
or the STNGUIDE file for information on formats available in
individual files.
REENTER DISPLAY FORMAT FOR ALL FILES (FILEDEFAULT):end

=> D IBIB ABS HIT 1-59

L122 ANSWER 1 OF 59     MEDLINE                  DUPLICATE 1
ACCESSION NUMBER:   2002178480     MEDLINE
DOCUMENT NUMBER:    21893335   PubMed ID: 11896118
TITLE:              Hepatic drug targeting: phase I evaluation of polymer-bound
                    doxorubicin.
AUTHOR:             Seymour Leonard W; Ferry David R; Anderson David;
                    Hesslewood Stuart; Julyan Peter J; Poyner Richard; Doran
                    Jayne; Young Annie M; Burtles Sally; Kerr David J
CORPORATE SOURCE:   Cancer Research UK Institute for Cancer Studies, University
                    of Birmingham, United Kingdom. (Cancer Research Campaign
                    Phase I/II Clinical Trials committee).
SOURCE:             JOURNAL OF CLINICAL ONCOLOGY, (2002 Mar 15) 20 (6) 1668-76.
                    Journal code: 8309333. ISSN: 0732-183X.
PUB. COUNTRY:       United States
DOCUMENT TYPE:      (CLINICAL TRIAL)
                    (CLINICAL TRIAL, PHASE I)
                    Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:           English
FILE SEGMENT:       Priority Journals
ENTRY MONTH:        200204
ENTRY DATE:         Entered STN: 20020326
```

combine Biosis answer sets

Remove duplicates from combined answer sets.

include "hit" in display terms to provide registry number of hit compounds - these databases will display the hit structure.

Last Updated on STN: 20020418
Entered Medline: 20020417

AB PURPOSE: Preclinical studies have shown good anticancer activity following targeting of a polymer bearing doxorubicin with galactosamine (PK2) to the liver. The present phase I study was devised to determine the toxicity, pharmacokinetic profile, and targeting capability of PK2. PATIENTS AND METHODS: Doxorubicin was linked via a lysosomally degradable tetrapeptide sequence to N-(2-hydroxypropyl)methacrylamide copolymers bearing galactosamine. Targeting, toxicity, and efficacy were evaluated in 31 patients with primary (n = 25) or metastatic (n = 6) liver cancer. Body distribution of the radiolabelled polymer conjugate was assessed using gamma-camera imaging and single-photon emission computed tomography. RESULTS: The polymer was administered by intravenous (i.v.) infusion over 1 hour, repeated every 3 weeks. Dose escalation proceeded from 20 to 160 mg/m(2) (doxorubicin equivalents), the maximum-tolerated dose, which was associated with severe fatigue, grade 4 neutropenia, and grade 3 mucositis. Twenty-four hours after administration, 16.9% +/- 3.9% of the administered dose of doxorubicin targeted to the liver and 3.3% +/- 5.6% of dose was delivered to tumor. Doxorubicin-polymer conjugate without galactosamine showed no targeting. Three hepatoma patients showed partial responses, with one in continuing partial remission 47 months after therapy. CONCLUSION: The recommended PK2 dose is 120 mg/m(2), administered every 3 weeks by IV infusion. Liver-specific doxorubicin delivery is achievable using galactosamine-modified polymers, and targeting is also seen in primary hepatocellular tumors.

CT Check Tags: Female; Human; Male; Support, Non-U.S. Gov't
Antineoplastic Agents: AD, administration & dosage
*Antineoplastic Agents: AE, adverse effects
***Antineoplastic Agents: PK, pharmacokinetics**
Area Under Curve
Chromatography, High Pressure Liquid
Doxorubicin: AD, administration & dosage
*Doxorubicin: AE, adverse effects
*Doxorubicin: AA, analogs & derivatives
Doxorubicin: CH, chemistry
*Doxorubicin: PK, pharmacokinetics
Drug Carriers
Galactosamine: AD, administration & dosage
Galactosamine: PK, pharmacokinetics
Gamma Cameras
Infusions, Intravenous
*Liver Neoplasms: DT, drug therapy
Polymethacrylic Acids: CH, chemistry
Tomography, Emission-Computed, Single-Photon
Treatment Outcome

RN 23214-92-8 (Doxorubicin); **7535-00-4 (Galactosamine)** < hit compound

L122 ANSWER 2 OF 59 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2002:474338 BIOSIS
DOCUMENT NUMBER: PREV200200474338
TITLE: Glucosamine pathway imaging using 99mTc-EC-deoxyglucose in comparison with 18F-FDG.
AUTHOR(S): Yang, D. J. (1); Macapinlac, H. A. (1); Yu, D. (1); Azhdarinia, A. (1); Kohanim, S. (1); Bryant, J. L. (1); Kim, E. E. (1); Podoloff, D. A. (1)
CORPORATE SOURCE: (1) University of Texas M.D. Anderson Cancer Center, Houston, TX USA
SOURCE: Journal of Nuclear Medicine, (May, 2002) Vol. 43, No. 5 Supplement, pp. 368P. http://jnm.snmjournals.org. print. Meeting Info.: 49th Annual Meeting of the Society of

Nuclear Medicine Los Angeles, CA, USA June 15-19, 2002
ISSN: 0161-5505.
DOCUMENT TYPE: Conference
LANGUAGE: English
IT Major Concepts
Methods and Techniques; Pharmacology; Radiation Biology; Tumor Biology
IT Diseases
tumor: neoplastic disease
IT Chemicals & Biochemicals
fluorine-18 FDG [fluorine-18 fluorodeoxyglucose]: diagnostic - drug, imaging agent; glucosamine pathway: imaging; glucosamine-6-phosphate; technetium-99m ethylenedicysteine-deoxyglucose: diagnostic - drug, imaging agent
IT Alternate Indexing
Neoplasms (MeSH)
RN 63503-12-8 (FLUORINE-18 FLUORODEOXYGLUCOSE)
3616-42-0 (GLUCOSAMINE-6-PHOSPHATE)

L122 ANSWER 3 OF 59 MEDLINE
ACCESSION NUMBER: 2002314753 MEDLINE
DOCUMENT NUMBER: 22051237 PubMed ID: 12056510
TITLE: TNF tolerance and cytotoxicity in the liver: the role of interleukin-1beta, inducible nitric oxide-synthase and heme oxygenase-1 in D-galactosamine-sensitized mice.
AUTHOR: Sass G; Koerber K; Tiegs G
CORPORATE SOURCE: Institute of Experimental and Clinical Pharmacology and Toxicology, University of Erlangen-Nuremberg, Germany.
SOURCE: INFLAMMATION RESEARCH, (2002 May) 51 (5) 229-35.
Journal code: 9508160. ISSN: 1023-3830.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200211
ENTRY DATE: Entered STN: 20020612
Last Updated on STN: 20021214
Entered Medline: 20021129

AB OBJECTIVE AND DESIGN: Pretreatment with tumor necrosis factor (TNF)-alpha induces tolerance towards itself in experimental liver injury. MATERIAL AND TREATMENT: To study mechanisms of TNF tolerance we used knockout mice for either TNF-receptor-2 (TNFR-2), inducible nitric oxide (NO)-synthase (iNOS) or caspase-1 (ICE) or inhibited heme oxygenase-1 (HO-1) by treatment with zinc-protoporphyrin 9. Liver damage was induced by administration of TNF to mice sensitized with D-galactosamine (GalN). Tolerance was induced by pretreatment with low doses of TNF. METHODS: Severity of liver injury was assessed by determination of plasma transaminases and apoptosis. Time courses of intra-hepatic iNOS, interleukin-1beta (IL-1beta) and HO-1 expression after TNF treatment were measured by reverse transcription polymerase chain reaction (RT-PCR). TNF-receptor-1 (TNFR-1) expression was determined by immunofluorescent staining. RESULTS: TNF-pretreatment did not affect TNFR-1 expression in the liver and resulted in time dependent up-regulation of iNOS, IL-1beta and HO-1. TNF- pretreated TNFR-2, iNOS or ICE knockout mice were as sensitive towards GalN/TNF as the wild type, while mice with impaired HO-1 activity were even more sensitive, but tolerance was inducible in all TNF-pretreated mice. CONCLUSIONS: TNF tolerance towards GalN/TNF treatment is mediated by TNFR-1. IL-1beta, iNOS and HO-1 neither mediated TNF-tolerance nor TNF cytotoxicity.
CT Check Tags: Animal; Male; Support, Non-U.S. Gov't
Antigens, CD: DE, drug effects

***Antineoplastic Agents: PD, pharmacology**
Antineoplastic Agents: TO, toxicity
DNA Fragmentation: DE, drug effects
Drug Tolerance
*Galaçtosamine: TO, toxicity
*Heme Oxygenase (Decyclizing): PH, physiology
*Hepatitis, Toxic: PA, pathology
Immunohistochemistry
*Interleukin-1: PH, physiology
Liver: DE, drug effects
*Liver: PA, pathology
Mice
Mice, Inbred BALB C
Mice, Inbred C57BL
Mice, Knockout
Microscopy, Confocal
*Nitric-Oxide Synthase: PH, physiology
RNA, Messenger: BI, biosynthesis
Receptors, Tumor Necrosis Factor: DE, drug effects
Reverse Transcriptase Polymerase Chain Reaction
*Tumor Necrosis Factor: PD, pharmacology
Tumor Necrosis Factor: TO, toxicity
RN **7535-00-4 (Galactosamine)**

L122 ANSWER 4 OF 59 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2002:192178 BIOSIS
DOCUMENT NUMBER: PREV200200192178
TITLE: Alkylating agents from sugars: Synthesis of chlorambucil derivatives carried by chiral glycosyl glycerols derived from D-glucosamine.
AUTHOR(S): Iglesias-Guerra, Fernando (1); Candela, Jose I.; Blanco, Eugenia; Alcudia, Felipe; Vega-Perez, Jose M.
CORPORATE SOURCE: (1) Departamento de Quimica Organica y Farmaceutica, Facultad de Farmacia, Universidad de Sevilla, E-41071, Sevilla: iglesias@fafar.us.es Spain
SOURCE: Chirality, (March, 2002) Vol. 14, No. 2-3, pp. 199-203. print.
ISSN: 0899-0042.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Chlorambucilamide derivatives involving chiral glycosyl glycerols derived from D-glucosamine were synthesized in good yield by coupling the chlorambucil moiety to the amino group of omega- amino-(omega-1)-hydroxyalkyl 2-acylamino-4,6-O-benzylidene-2-deoxy-beta-D-glucopyranosides, and subsequent hydrolysis of the benzylidene group. The starting material was easily available from 2-acetamido-2-deoxy-D-glucose. The bonding of 2,3,4,6-tetra-O-pivaloyl-beta-D-galactopyranosylamine to chlorambucil by formation of an amide function is also described.
IT Major Concepts
Biochemistry and Molecular Biophysics; Methods and Techniques; Pharmacology
IT Diseases
cancer: neoplastic disease; malignant metastases: neoplastic disease
IT Chemicals & Biochemicals
2,3,4,5-tetra-O-pivaloyl-beta-D-galactopyranosylamine: bonding; 2-acetamido-2-deoxy-D-glucose; D-glucosamine; alkylating agents; amino-sugars; antitumor agents: antineoplastic - drug, preparation; benzylidene group; chiral glycosyl glycerols; chlorambucil: moiety; chlorambucil derivatives: synthesis; chlorambucilamide derivatives: synthesis; hydrolysis: synthetic method; omega-amino-(omega-1)-

hydroxyalkyl 2-acylamino-4,6-O-benzylidene-2-deoxy-beta-D-glucopyranodises; sugars
IT Alternate Indexing
Neoplasms (MeSH)
RN 7512-17-6 (2-ACETAMIDO-2-DEOXY-D-GLUCOSE)
3416-24-8 (D-GLUCOSAMINE)
305-03-3 (CHLORAMBUCIL)
305-03-3D (CHLORAMBUCIL)
57-50-1 (SUGARS)

L122 ANSWER 5 OF 59 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2001300282 EMBASE
TITLE: Angiogenesis: A therapeutic target in arthritis.
AUTHOR: Walsh D.A.; Haywood L.
CORPORATE SOURCE: D.A. Walsh, Academic Rheumatology, University of Nottingham, City Hospital, Hucknall Road, Nottingham NG5 1PB, United Kingdom. David.Walsh@nottingham.ac.uk
SOURCE: Current Opinion in Investigational Drugs, (2001) 2/8 (1054-1063).
Refs: 109
ISSN: 0967-8298 CODEN: CIDREE
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 031 Arthritis and Rheumatism
037 Drug Literature Index
030 Pharmacology
016 Cancer
005 General Pathology and Pathological Anatomy
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
AB A variety of pharmacological strategies are being subjected to clinical trial to inhibit neovascularization of solid tumors. Increased angiogenesis is also a key component of synovitis and bone modeling in arthritis. Molecular mechanisms and pathological consequences of blood vessel growth in arthritis are now being elucidated. Preclinical studies of angiogenesis inhibitors in animal models of inflammatory arthritis support the hypothesis that inhibition of neovascularization may reduce inflammation and joint damage. Clinical data are consistent with these models being predictive of efficacy in rheumatoid arthritis. However, controlled studies of specific anti-angiogenic agents in human arthritis remain limited. Further studies are required to demonstrate that pharmacological agents can effectively inhibit articular angiogenesis, and ameliorate inflammation and subsequent joint damage. Potential toxicity of angiogenesis inhibitors in reproduction, growth and development and wound repair may be circumvented by short-term or local application, or by targeting molecular mechanisms that are specific to pathological rather than physiological angiogenesis.
CT Medical Descriptors:
*arthritis: ET, etiology
*arthritis: DT, drug therapy
*angiogenesis
human
clinical trial
nonhuman
drug targeting
treatment planning
pharmaceutical engineering
solid tumor: ET, etiology
synovitis: ET, etiology

bone remodeling
molecular dynamics
drug screening
disease model
inflammatory disease: ET, etiology
inflammatory disease: DT, drug therapy
inhibition kinetics
drug mechanism
arthropathy
inflammation
drug efficacy
rheumatoid arthritis: DT, drug therapy
articular cartilage
reproductive toxicity: SI, side effect
growth, development and aging disorders: SI, side effect
wound healing impairment: SI, side effect
drug exposure
drug specificity
ossification
ankylosing spondylitis: DT, drug therapy
osteoarthritis: DT, drug therapy
receptor down regulation
review
Drug Descriptors:
***angiogenesis inhibitor: DT, drug therapy**
*angiogenesis inhibitor: DV, drug development
*angiogenesis inhibitor: PD, pharmacology
*angiogenesis inhibitor: AE, adverse drug reaction
*angiogenesis inhibitor: CT, clinical trial
*angiogenesis inhibitor: PK, pharmacokinetics
*antirheumatic agent: DT, drug therapy
*antirheumatic agent: DV, drug development
*antirheumatic agent: PD, pharmacology
*antirheumatic agent: AE, adverse drug reaction
*antirheumatic agent: CT, clinical trial
*antirheumatic agent: PK, pharmacokinetics
vascular cell adhesion molecule 1: EC, endogenous compound
endothelial leukocyte adhesion molecule 1: EC, endogenous compound
angiogenesis factor inhibitor derivative: DT, drug therapy
angiogenesis factor inhibitor derivative: PK, pharmacokinetics
angiogenesis factor inhibitor derivative: PD, pharmacology
adhesion molecule inhibitor: DT, drug therapy
adhesion molecule inhibitor: PK, pharmacokinetics
adhesion molecule inhibitor: PD, pharmacology
protein tyrosine kinase inhibitor: DT, drug therapy
protein tyrosine kinase inhibitor: PK, pharmacokinetics
protein tyrosine kinase inhibitor: PD, pharmacology
vasculotropin inhibitor: DT, drug therapy
vasculotropin inhibitor: PK, pharmacokinetics
vasculotropin inhibitor: PD, pharmacology
mitogen activated protein kinase inhibitor: DT, drug therapy
mitogen activated protein kinase inhibitor: PK, pharmacokinetics
mitogen activated protein kinase inhibitor: PD, pharmacology
peroxisome proliferator activated receptor gamma: EC, endogenous compound
ligand: DT, drug therapy
ligand: PK, pharmacokinetics
ligand: PD, pharmacology
endostatin: DT, drug therapy
endostatin: PK, pharmacokinetics
endostatin: PD, pharmacology

angiostatin: DT, drug therapy
angiostatin: PK, pharmacokinetics
angiostatin: PD, pharmacology
sialic acid derivative: DT, drug therapy
sialic acid derivative: PK, pharmacokinetics
sialic acid derivative: PD, pharmacology
microtubule inhibitor: DT, drug therapy
microtubule inhibitor: PK, pharmacokinetics
microtubule inhibitor: PD, pharmacology
suramin: DT, drug therapy
suramin: PK, pharmacokinetics
suramin: PD, pharmacology
mannosamine: DT, drug therapy
mannosamine: PK, pharmacokinetics
mannosamine: PD, pharmacology
hydroxymethylglutaryl coenzyme A reductase inhibitor: DT, drug therapy
hydroxymethylglutaryl coenzyme A reductase inhibitor: PK, pharmacokinetics
hydroxymethylglutaryl coenzyme A reductase inhibitor: PD, pharmacology
bisphosphonic acid derivative: DT, drug therapy
bisphosphonic acid derivative: PK, pharmacokinetics
bisphosphonic acid derivative: PD, pharmacology
combretastatin: DT, drug therapy
combretastatin: PK, pharmacokinetics
combretastatin: PD, pharmacology
isocoumarin derivative: DT, drug therapy
isocoumarin derivative: PK, pharmacokinetics
isocoumarin derivative: PD, pharmacology
dextrin: DT, drug therapy
dextrin: PK, pharmacokinetics
dextrin: PD, pharmacology
endoglin: DT, drug therapy
endoglin: PK, pharmacokinetics
endoglin: PD, pharmacology
gamma interferon: DT, drug therapy
gamma interferon: PK, pharmacokinetics
gamma interferon: PD, pharmacology
leukemia inhibitory factor: DT, drug therapy
leukemia inhibitory factor: PK, pharmacokinetics
leukemia inhibitory factor: PD, pharmacology
tissue inhibitor of metalloproteinase 1: DT, drug therapy
tissue inhibitor of metalloproteinase 1: PK, pharmacokinetics
tissue inhibitor of metalloproteinase 1: PD, pharmacology
tissue inhibitor of metalloproteinase 2: DT, drug therapy
tissue inhibitor of metalloproteinase 2: PK, pharmacokinetics
tissue inhibitor of metalloproteinase 2: PD, pharmacology
angiopoietin 2: DT, drug therapy
angiopoietin 2: PD, pharmacology
angiopoietin 2: PK, pharmacokinetics
transforming growth factor beta: DT, drug therapy
transforming growth factor beta: PD, pharmacology
transforming growth factor beta: PK, pharmacokinetics
unindexed drug
unclassified drug
RN (endothelial leukocyte adhesion molecule 1) 128875-25-2; (endostatin) 187888-07-9; (angiostatin) 172642-30-7, 86090-08-6; (suramin) 129-46-4, 145-63-1; (mannosamine) **14307-02-9, 2636-92-2;** (combretastatin) 82855-09-2, 89064-44-8; (dextrin) 9004-53-9; (gamma interferon) 82115-62-6; (tissue inhibitor of metalloproteinase 1) 140208-24-8; (tissue inhibitor of metalloproteinase 2) 124861-55-8; (angiopoietin 2) 194368-66-6

L122 ANSWER 6 OF 59 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2001437633 EMBASE
TITLE: New therapeutic approaches in rheumatology.
AUTHOR: Machacek S.
CORPORATE SOURCE: Dr. S. Machacek, Vyzlovska 2251, Prague 10, 11000, Czech Republic
SOURCE: Drug News and Perspectives, (2001) 14/7 (428-435).
ISSN: 0214-0934 CODEN: DNPEED
COUNTRY: Spain
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 031 Arthritis and Rheumatism
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English

AB This year's European League Against Rheumatism meeting, held in Prague, June 13-16, 2001, was attended by approximately 8,200 registered scientists and physicians. The meeting covered a broad spectrum of topics on rheumatic diseases, including pathogenesis, diagnostics and treatment. New therapeutic approaches to systemic diseases were the highlight of this congress. .COPYRGT. 2001 Prous Science. All rights reserved.

CT Medical Descriptors:
*rheumatology
*rheumatic disease: DI, diagnosis
*rheumatic disease: DT, drug therapy
*rheumatic disease: ET, etiology
*osteoporosis: DT, drug therapy
Europe
clinical practice
pathogenesis
early diagnosis
fibromyalgia: DT, drug therapy
osteoarthritis: DT, drug therapy
human
conference paper
Drug Descriptors:
*nonsteroid antiinflammatory agent: DT, drug therapy
*corticosteroid: DT, drug therapy
*infliximab: DT, drug therapy
*methotrexate: CB, drug combination
***methotrexate: DT, drug therapy**
*etanercept: CB, drug combination
*etanercept: DT, drug therapy
*salazosulfapyridine: DT, drug therapy
*recombinant interleukin 1 receptor blocking agent: DT, drug therapy
alendronic acid: DT, drug therapy
ibandronic acid: DT, drug therapy
risedronic acid: DT, drug therapy
hyaluronic acid: DT, drug therapy
diacetylrhein: DT, drug therapy
glucosamine sulfate: DT, drug therapy
alfacalcidol: DT, drug therapy
tramadol: DT, drug therapy
sertraline: DT, drug therapy

RN (infliximab) 170277-31-3; (methotrexate) 15475-56-6, 59-05-2, 7413-34-5; (etanercept) 185243-69-0, 200013-86-1; (salazosulfapyridine) 599-79-1; (alendronic acid) 66376-36-1; (ibandronic acid) 114084-78-5, 138844-81-2, 138926-19-9; (risedronic acid) 105462-24-6, 122458-82-6; (hyaluronic acid) 31799-91-4, 9004-61-9, 9067-32-7; (diacetylrhein) 13739-02-1; (glucosamine sulfate) **29031-19-4**; (alfacalcidol) 41294-56-8; (tramadol)

27203-92-5, 36282-47-0; (sertraline) 79617-96-2

L122 ANSWER 7 OF 59 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2001228459 EMBASE
TITLE: Protective effect of OK-432 on mice against endotoxemia and infection with Pseudomonas aeruginosa and Salmonella enteritidis.
AUTHOR: Hashimoto M.; Kirikae F.; Toyooka K.; Kaneko A.; Yamasu H.; Iwai H.; Nakano M.; Kirikae T.
CORPORATE SOURCE: Dr. T. Kirikae, Department of Infectious Diseases, Research Institute, International Medical Ctr. of Japan, Toyama 1-21-1, Shinjuku-ku, Tokyo 162-8655, Japan. tkirikae@ri.imcj.go.jp
SOURCE: Microbiology and Immunology, (2001) 45/6 (425-432). Refs: 40
ISSN: 0385-5600 CODEN: MIIMDV
COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
026 Immunology, Serology and Transplantation
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English

AB OK-432 has been used clinically as a biological response modifier for cancer therapy. We investigated here the protective effects of OK-432 against endotoxic shock and infectious death caused by Pseudomonas aeruginosa and Salmonella enteritidis in mice and proposed a possible mechanism. Pretreatment of OK-432 reduced the lethality of lipopolysaccharide (LPS)-induced endotoxic shock in D-(+)-galactosamine-sensitized C3H/HeN mice. OK-432 did not affect the TNF.alpha. production in blood, but it did decrease the susceptibility to TNF.alpha.. Furthermore, an acceleration of LPS clearance from blood was detected. The pretreatment of OK-432 also decreased the lethality of mice in bacterial infection caused by P. aeruginosa and S. enteritidis. The rapid decrease of the viable bacteria from the circulating blood and in spleen and liver in mice was observed in a manner similar to LPS clearance. These findings indicate that the protective effect of OK.432 against the endotoxemia and bacteremia may depend on an up-regulation of clearance of LPS and bacteria and the augmented resistance to TNF.alpha..

CT Medical Descriptors:
*endotoxemia: DT, drug therapy
*endotoxemia: PC, prevention
*Pseudomonas aeruginosa
*Salmonella enteritidis
*infection: PC, prevention
cell protection
lethality
cytokine production
immunomodulation
LD 50
infection risk
spleen
liver clearance
dose time effect relation
survival
bacteremia
septic shock: DT, drug therapy
septic shock: PC, prevention
nonhuman
male

female
mouse
animal model
controlled study
animal tissue
animal cell
article
Drug Descriptors:
***picibanil: DT, drug therapy**
*picibanil: PD, pharmacology
*picibanil: IP, intraperitoneal drug administration
*biological response modifier
tumor necrosis factor alpha: EC, endogenous compound
bacterium lipopolysaccharide
galactosamine
RN (picibanil) 39325-01-4; (galactosamine) **7535-00-4**

L122 ANSWER 8 OF 59 MEDLINE
ACCESSION NUMBER: 2001443725 MEDLINE
DOCUMENT NUMBER: 21382305 PubMed ID: 11489490
TITLE: Polymer-drug conjugates, PDEPT and PELT: basic principles for design and transfer from the laboratory to clinic.
AUTHOR: Duncan R; Gac-Breton S; Keane R; Musila R; Sat Y N; Satchi R; Searle F
CORPORATE SOURCE: Centre for Polymer Therapeutics, Welsh School of Pharmacy, Cardiff University, Redwood Building, King Edward VII Avenue, Cardiff CF10 3XF, Wales, UK.. duncanr@cf.ac.uk
SOURCE: JOURNAL OF CONTROLLED RELEASE, (2001 Jul 6) 74 (1-3) 135-46.
Journal code: 8607908. ISSN: 0168-3659.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200112
ENTRY DATE: Entered STN: 20010813
Last Updated on STN: 20020121
Entered Medline: 20011204

AB There are now at least seven polymer-drug conjugates that have entered phase I/II clinical trial as anticancer agents. These include N-(2-hydroxypropyl)methacrylamide (HPMA) copolymer-doxorubicin (PK1, FCE28068), HPMA copolymer-paclitaxel (PNU 166945), HPMA copolymer-camptothecin, PEG-camptothecin, polyglutamic acid-paclitaxel, an HPMA copolymer-platinate (AP5280) and also an HPMA copolymer-doxorubicin conjugate bearing additionally galactosamine (PK2, FCE28069). The galactosamine is used as a means to target the conjugate to liver for the treatment of primary and secondary liver cancer. Promising early clinical results with lysosomotropic conjugates has stimulated significant interest in this field. Ongoing research is developing (1) conjugates containing drugs that could otherwise not progress due to poor solubility or uncontrollable toxicity; (2) conjugates of agents directed against novel targets; and (3) two-step combinations such as polymer-directed enzyme prodrug therapy (PDEPT) and polymer-enzyme liposome therapy (PELT) that can cause explosive liberation of drug from either polymeric prodrugs or liposomes within the tumour interstitium. Moreover, bioresponsive polymer-based constructs able to promote endosomal escape and thus intracytoplasmic delivery of macromolecular drugs (peptides, proteins and oligonucleotides) are also under study.

CT Check Tags: Animal
Acrylamides: AD, administration & dosage

Acrylamides: PD, pharmacology
Antibiotics, Anthracycline: AD, administration & dosage
Antibiotics, Anthracycline: CH, chemistry
Antibiotics, Anthracycline: TU, therapeutic use
***Antineoplastic Agents: AD, administration & dosage**
Antineoplastic Agents: PD, pharmacology
Doxorubicin: AD, administration & dosage
Doxorubicin: CH, chemistry
Doxorubicin: PD, pharmacology
Doxorubicin: TU, therapeutic use
Drug Carriers
*Drug Delivery Systems
Excipients
Galactosamine: AD, administration & dosage
Galactosamine: PD, pharmacology
Liposomes
Methacrylates
Mice
Neoplasms, Experimental: DT, drug therapy
Organoplatinum Compounds: AD, administration & dosage
Organoplatinum Compounds: PD, pharmacology
*Polymers: CH, chemistry
*Prodrugs: AD, administration & dosage
RN 23214-92-8 (Doxorubicin); 27813-02-1 (hydroxypropyl methacrylate); **7535-00-4 (Galactosamine)**

L122 ANSWER 9 OF 59 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2001:366048 BIOSIS
DOCUMENT NUMBER: PREV200100366048
TITLE: Progress in Drug Research.
AUTHOR(S): Jucker, Ernst (1)
CORPORATE SOURCE: (1) Steinweg 28, CH-4107, Ettingen: jucker.pdr@bluewin.ch Switzerland
SOURCE: Jucker, Ernst. Progress in Drug Research, (2000) Vol. 55, pp. i-viii, 1-334. Progress in Drug Research. print. Publisher: Birkhaeuser Publishing Ltd. CH-4010, Basel, Switzerland. ISSN: 0071-786X. ISBN: 3-7643-6193-X (cloth).
DOCUMENT TYPE: Book
LANGUAGE: English
SUMMARY LANGUAGE: English
AB This volume contains 7 separately authored articles on the latest information in drug research. It also contains a title index and an author and paper index for Volumes 1-55 of this series. A subject index and bibliographical references are included.
IT Major Concepts
Pharmacology
IT Parts, Structures, & Systems of Organisms
cell: proliferation
IT Diseases
hepatitis C: digestive system disease, viral disease; osteoarthritis: joint disease; prostate cancer: neoplastic disease, reproductive system disease/male, urologic disease
IT Chemicals & Biochemicals
androgen receptor; **antineoplastic agent**; antiviral agent; cardiotonic agent: cardiovascular agent, quantitative structure-activity relationships; chondroitin sulfate: antiarthritic - drug; glucosamine sulfate: antiarthritic - drug; morphine: growth regulator
IT Alternate Indexing

Hepatitis C (MeSH); Osteoarthritis (MeSH); Prostatic Neoplasms (MeSH)
RN 9007-28-7 (CHONDROITIN SULFATE)
29031-19-4 (GLUCOSAMINE SULFATE)
57-27-2 (MORPHINE)

L122 ANSWER 10 OF 59 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2000179854 EMBASE
TITLE: Protective effects of gram-positive bacterial components against endotoxic shock in mice.
AUTHOR: Kirikae T.; Suda Y.; Tamura H.; Yamasu H.; Kirikae F.; Iwai H.; Hashimoto M.; Kusumoto S.; Nakano M.
CORPORATE SOURCE: T. Kirikae, Department of Infectious Diseases, Tropical Medicine, Intl. Medical Center of Japan, Tokyo 162-8655, Japan
SOURCE: Japanese Journal of Infectious Diseases, (2000) 53/1 (34). Refs: 6
ISSN: 1344-6304 CODEN: JJIDFE
COUNTRY: Japan
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 004 Microbiology
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
CT Medical Descriptors:
*septic shock: DT, drug therapy
*septic shock: PC, prevention
*Streptococcus pyogenes
Gram positive bacterium
Japan
Pseudomonas aeruginosa
Gram negative infection
bacterial count
bacterium culture
drug mechanism
Enterococcus hirae
drug effect
nonhuman
mouse
animal experiment
animal model
controlled study
conference paper
Drug Descriptors:
***picibanil: DT, drug therapy**
*picibanil: PD, pharmacology
*biological response modifier: DT, drug therapy
*biological response modifier: PD, pharmacology
galactosamine
lipoteichoic acid: EC, endogenous compound
glycoconjugate
endotoxin
ceftazidime
tumor necrosis factor
lipopolysaccharide
RN (picibanil) 39325-01-4; (galactosamine) **7535-00-4**; (lipoteichoic acid) 56411-57-5; (ceftazidime) 72558-82-8

L122 ANSWER 11 OF 59 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2000046402 EMBASE
TITLE: [Diagnosis of lysosomal storage diseases].

DIAGNOSTIEK VAN LYSOSOMALE STAPELINGSZIEKTEN.

AUTHOR: De Jong J.G.N.; Wevers R.; Van den Berg C.J.M.; Liebrand-van Sambeek M.L.F.; Van Rens A.A.E.T.; Roelofs H.G.M.

CORPORATE SOURCE: Dr. J.G.N. De Jong, Academisch Ziekenhuis Nijmegen, 319 Lab. Kindergeneeskunde/Neurol., Reinier Postlaan 4, 6525 GC Nijmegen, Netherlands. j.dejong@ckslkn.azn.nl

SOURCE: Nederlands Tijdschrift voor de Klinische Chemie, (2000) 25/1 (13-27).
Refs: 39
ISSN: 1380-3689 CODEN: NTKCFX

COUNTRY: Netherlands
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: German
SUMMARY LANGUAGE: English; German

AB Most of the lysosomal storage diseases underlies a deficiency of one of the lysosomal enzymes involved in the degradation of macromolecules to their monomolecular building blocks. On basis of clinical symptoms and storage products three main groups can be recognized, the sphingolipidoses, oligosaccharidoses and mucopolysaccharidoses. Mucolipidoses II and III are caused by a defect in the processing of the lysosomal enzymes, leading to a deficiency of several enzymes together. Lysosomal storage can also be due to a defect in the transport, for example for neuraminic acid, over the lysosomal membrane. For some of the neuronal ceroid lipofuscinoses it has been shown now that they belong to the group of lysosomal storage diseases. Clinical symptoms for the group of lysosomal storage diseases are heterogeneous. The most characteristic are decrease in mental and or motoric development and hepato- and or splenomegaly. Screening in urine is possible for 10 oligosaccharidoses by analysis of oligosaccharides and for the mucopolysaccharidoses by measurement of glycosaminoglycan content in urine. A defect in the transport of neuraminic acid can be detected by measurement of this compound in the urine. When an abnormal oligosaccharide pattern is found or the glycosaminoglycan excretion is increased the concerning lysosomal enzymes are measured in leukocytes, isolated from a blood sample to confirm or exclude the defect. Screening in urine is not possible for most of the sphingolipidoses and for the neuronal ceroid lipofuscinoses. For the sphingolipidoses and some of the neuronal ceroid lipofuscinoses diagnosis can be made by direct measurement of the various enzymes in leukocytes and/or fibroblasts.

CT Medical Descriptors:
*lysosome storage disease: CN, congenital disorder
*lysosome storage disease: DI, diagnosis
enzyme deficiency
enzyme degradation
mucolipidosis type 2: CN, congenital disorder
mucolipidosis type 2: DI, diagnosis
mucolipidosis type 3: CN, congenital disorder
mucolipidosis type 3: DI, diagnosis
lipidosis: CN, congenital disorder
lipidosis: DI, diagnosis
neuronal ceroid lipofuscinosis: CN, congenital disorder
neuronal ceroid lipofuscinosis: DI, diagnosis
human
review
Drug Descriptors:
*neuraminic acid: EC, endogenous compound
*lipofuscin: EC, endogenous compound
*oligosaccharide: EC, endogenous compound

*glycosaminoglycan: EC, endogenous compound
RN (neuraminic acid) **114-04-5**

L122 ANSWER 12 OF 59 MEDLINE
ACCESSION NUMBER: 1999364592 MEDLINE
DOCUMENT NUMBER: 99364592 PubMed ID: 10437881
TITLE: Glycoscience moves from the laboratory to the clinic.
AUTHOR: Rowe P M
SOURCE: LANCET, (1999 Jul 31) 354 (9176) 402.
Journal code: 2985213R. ISSN: 0140-6736.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: News Announcement
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals; AIDS
ENTRY MONTH: 199908
ENTRY DATE: Entered STN: 19990820
Last Updated on STN: 19990820
Entered Medline: 19990812

CT Check Tags: Human
1-Deoxynojirimycin: CH, chemistry
*1-Deoxynojirimycin: TU, therapeutic use
Anti-HIV Agents: CH, chemistry
*Anti-HIV Agents: TU, therapeutic use
Antibiotics: CH, chemistry
*Antibiotics: TU, therapeutic use
Antineoplastic Agents: CH, chemistry
***Antineoplastic Agents: TU, therapeutic use**
Antiviral Agents: CH, chemistry
*Antiviral Agents: TU, therapeutic use
Enzyme Inhibitors: CH, chemistry
*Enzyme Inhibitors: TU, therapeutic use
*Glucosamine: AA, analogs & derivatives
Glucosamine: CH, chemistry
Glucosamine: TU, therapeutic use
HIV-1: DE, drug effects
Structure-Activity Relationship
Swainsonine: CH, chemistry
*Swainsonine: TU, therapeutic use
RN 15218-38-9 (nojirimycin); 19130-96-2 (1-Deoxynojirimycin); **3416-24-8 (Glucosamine)**; 72741-87-8 (Swainsonine)

L122 ANSWER 13 OF 59 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1999:286896 BIOSIS
DOCUMENT NUMBER: PREV199900286896
TITLE: Review: Occurrence of sialic acids in healthy humans and different disorders.
AUTHOR(S): Sillanaukee, P. (1); Ponnio, M.; Jaaskelainen, I. P.
CORPORATE SOURCE: (1) R and D, Pharmacia and Upjohn Diagnostics AB, S-751 82, Uppsala Sweden
SOURCE: European Journal of Clinical Investigation, (May, 1999) Vol. 29, No. 5, pp. 413-425.
ISSN: 0014-2972.
DOCUMENT TYPE: General Review
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Sialic acid (SA), N-acetylated derivatives of neuraminic acid, play a central role in the biomedical functioning of humans. The normal range of total sialic acid (TSA) level in serum/plasma is 1.58-2.22 mmol L-1, the free form of SA only constituting 0.5-3 mumol L-1 and the lipid-associated (LSA) forms 10-50 mumol L-1. Notably, considerably higher amounts of free

SA are found in urine than in serum/plasma (approximately 50% of the total SA). In inherited SA storage diseases such as Salla's disease, SA levels are elevated many times over, and their determination during clinical investigation is well established. Furthermore, a number of reports describe elevated SA levels in various other diseases, tentatively suggesting broader clinical utility for SA markers. Increased SA concentrations have been reported during inflammatory processes, probably resulting from increased levels of richly sialylated acute-phase glycoproteins. A connection between increased SA levels and elevated stroke and cardiovascular mortality risk has also been reported. In addition, SA levels are slightly increased in cancer, positively correlating with the degree of metastasis, as well as in alcohol abuse, diabetes, chronic renal failure and chronic glomerulonephritis. Several different mechanisms are assumed to underlie the elevated SA concentrations in these disorders. The apparent non-specificity of SA to a given disease limits the potential clinical usefulness of SA determination. In addition, some non-pathological factors, such as aging, pregnancy and smoking, may cause changes in SA concentrations. The absolute increases in SA levels are also rather small (save those in inherited SA storage disorders); this further limits the clinical potential of SA as a marker. Tentatively, SA markers might serve as adjuncts, when combined with other markers, in disease screening, disease progression follow-up, and in the monitoring of treatment response. To become clinically useful, however, the existing SA determination assays need to be considerably refined to reduce interferences, to be specific for certain SA forms, and to be more easy to use.

IT Major Concepts
Clinical Chemistry (Allied Medical Sciences); Human Medicine (Medical Sciences)

IT Diseases
alcohol abuse: behavioral and mental disorders, toxicity; cancer: metastasis, neoplastic disease; chronic glomerulonephritis: urologic disease; chronic renal failure: urologic disease; coronary heart disease: heart disease; diabetes: endocrine disease/pancreas, metabolic disease; stroke: nervous system disease, vascular disease; Salla's disease: metabolic disease

IT Chemicals & Biochemicals
neuraminic acid; sialic acids: disease marker, reference range, plasma, urine, serum

IT Alternate Indexing
Alcoholism (MeSH); Cerebrovascular Disorders (MeSH); Coronary Disease (MeSH); Diabetes Mellitus (MeSH); Glomerulonephritis (MeSH); Kidney Failure, Chronic (MeSH); **Neoplasms (MeSH)**

RN 131-48-6D (SIALIC ACIDS)
114-04-5 (NEURAMINIC ACID)
64-17-5 (ALCOHOL)

L122 ANSWER 14 OF 59 MEDLINE
ACCESSION NUMBER: 2000012675 MEDLINE
DOCUMENT NUMBER: 20012675 PubMed ID: 10547184
TITLE: Involvement of oxidative DNA damage and apoptosis in antitumor actions of aminosugars.
AUTHOR: Hiraku Y; Kawanishi S
CORPORATE SOURCE: Department of Hygiene, Mie University School of Medicine, Tsu, Japan.
SOURCE: FREE RADICAL RESEARCH, (1999 Nov) 31 (5) 389-403. Journal code: 9423872. ISSN: 1071-5762.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English

FILE SEGMENT: Priority Journals
ENTRY MONTH: 199911
ENTRY DATE: Entered STN: 20000111
Last Updated on STN: 20000111
Entered Medline: 19991119

AB We investigated the mechanisms of apoptosis and DNA damage induced by aminosugars in relation to their antitumor actions. The order of cytotoxic effects of aminosugars was D-mannosamine (ManN) >> D-galactosamine (GalN) > D-glucosamine (GlcN). A comparison of the frequency of apoptotic cells showed the same order. DNA ladders were formed by only ManN and the formation of DNA ladders was inhibited by a caspase inhibitor. Pulsed-field gel electrophoresis showed that ManN caused cellular DNA cleavage at a lower concentration than those causing apoptosis. Cellular DNA cleavage was inhibited by catalase and enhanced by a catalase inhibitor. Flow cytometry showed that ManN enhanced the production of intracellular peroxides. These results suggest that ManN-induced apoptosis is preceded by H2O2-mediated DNA damage. The order of the extent of damage to 32P-labeled DNA fragments by aminosugars plus Cu(II) was ManN >> GalN > GlcN. The DNA damage was inhibited by catalase and bathocuproine, suggesting that H2O2 reacts with Cu(I) to form the metal-peroxide complex capable of causing DNA damage. Two mechanisms of H2O2 generation from aminosugars were proposed: one is the major pathway to form a dioxo compound and NH4+; the other is the minor pathway to form a pyrazine derivative through the condensation of two molecules of an aminosugar. The order of reactivity to generate these products was ManN >> GalN > GlcN. On the basis of these results, it is concluded that aminosugars, especially ManN, produce H2O2 to cause DNA damage, which mediates apoptosis resulting in tumor growth inhibition.

CT Check Tags: Comparative Study; Human; Support, Non-U.S. Gov't
*Amino Sugars: PD, pharmacology
***Antineoplastic Agents: PD, pharmacology**
*Apoptosis: DE, drug effects
Caspases: AI, antagonists & inhibitors
Caspases: PD, pharmacology
Cell Survival: DE, drug effects
Copper
*DNA Damage: DE, drug effects
DNA Fragmentation
Enzyme Inhibitors: PD, pharmacology
Free Radical Scavengers
Galactosamine: PD, pharmacology
Glucosamine: PD, pharmacology
Hexosamines: PD, pharmacology
Hydrogen Peroxide: ME, metabolism
Hydrogen Peroxide: PD, pharmacology
Oxidation-Reduction
Phenanthrolines: PD, pharmacology
Tumor Cells, Cultured

RN **2636-92-2 (mannosamine)**; **3416-24-8 (Glucosamine)**; 4733-39-5 (bathocuproine); 7440-50-8 (Copper); **7535-00-4 (Galactosamine)**; 7722-84-1 (Hydrogen Peroxide)

L122 ANSWER 15 OF 59 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 1999192562 EMBASE
TITLE: Evaluation of effectiveness of glucocorticoid treatment using a rat acute hepatic failure model.
AUTHOR: Sato A.; Takikawa Y.; Sato S.; Suzuki K.
CORPORATE SOURCE: A. Sato, Iwate Medical University, First Dept. of Internal Medicine, Morioka, Japan
SOURCE: Acta Hepatologica Japonica, (1999) 40/4 (217-226).

Refs: 28
ISSN: 0451-4203 CODEN: KNZOAU
COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 030 Pharmacology
037 Drug Literature Index
048 Gastroenterology
LANGUAGE: Japanese
SUMMARY LANGUAGE: English; Japanese

AB We studied the effectiveness, mechanism of action and potential for clinical application of glucocorticoids using an acute hepatic failure model. Two hundred milligrams per kilogram of D-galactosamine and 10 .mu.g/kg of lipopolysaccharide were injected via the portal vein of 9-week-old Wistar rats to produce hepatic failure, and methylprednisolone (20 mg/kg) was injected via the portal vein for treatment. In the untreated group, increases in the serum levels of tumor necrosis factor .alpha. (TNF-.alpha.) and interleukin-8 (IL-8), and hepatic cell apoptosis peaking at 3 hrs, 6 hrs and 12 hrs, respectively, from the injection of GalN/LPS were observed. Furthermore, a marked increase in the serum concentrations of transaminases and T. Bil, as well as massive hepatic cell death were observed 24 hrs after the injection of GalN/LPS. In the concomitantly treated group, the increase in serum levels of TNF-.alpha. was significantly inhibited ($p < 0.05$), and apoptosis as well as hepatic failure, which developed 24 hrs after the injection was suppressed. However, in the delayed treatment group, suppression of neither hepatic damage nor massive hepatic cell death was evident. From the above results, it has been clarified that glucocorticoids inhibit both TNF-.alpha. production and the development of hepatic cell damage. However, the effect was not obvious when glucocorticoids were administered after the TNF-.alpha. peak. Therefore, the timing of glucocorticoid administration is an important factor that must be considered in the clinical application of glucocorticoids.

CT Medical Descriptors:
*corticosteroid therapy
*liver failure: DI, diagnosis
*liver failure: DT, drug therapy
drug efficacy
drug mechanism
cytokine production
apoptosis
dose time effect relation
aminotransferase blood level
bilirubin blood level
chronotherapy
liver protection
liver histology
nonhuman
rat
animal experiment
animal model
controlled study
animal tissue
intravenous drug administration
article
Drug Descriptors:
*glucocorticoid: DV, drug development
*glucocorticoid: DT, drug therapy
*glucocorticoid: PD, pharmacology
*methylprednisolone: DV, drug development
***methylprednisolone: DT, drug therapy**

*methylprednisolone: PD, pharmacology
galactosamine
lipopolysaccharide
tumor necrosis factor alpha: EC, endogenous compound
interleukin 8: EC, endogenous compound
aspartate aminotransferase: EC, endogenous compound
alanine aminotransferase: EC, endogenous compound
bilirubin: EC, endogenous compound
RN (methylprednisolone) 6923-42-8, 83-43-2; (galactosamine) **7535-00-4** ; (interleukin 8) 114308-91-7; (aspartate aminotransferase) 9000-97-9; (alanine aminotransferase) 9000-86-6, 9014-30-6; (bilirubin) 18422-02-1, 635-65-4

L122 ANSWER 16 OF 59 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 1999195845 EMBASE
TITLE: Differences in metabolism of 5-fluorouracil and 5-fluorouridine and regulation by glucosamine in human colon cancer multicell tumor spheroids.
AUTHOR: Chen T.-B.; Bajzer Z.; Macura S.; Vuk-Pavlovic S.
CORPORATE SOURCE: S. Vuk-Pavlovic, Guggenheim 1311A, Mayo Clinic, Rochester, MN 55905, United States
SOURCE: NMR in Biomedicine, (1999) 12/3 (157-167).
Refs: 22
ISSN: 0952-3480 CODEN: NMRBEF
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 014 Radiology
016 Cancer
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Glucosamine (GlcN) modulates fluoropyrimidine metabolism and enhances cytotoxicity of 5-fluorouridine (FUrd), but not of 5-fluorouracil (FUra), in human tumor models. To elucidate the underlying metabolic differences between FUra and FUrd, by the use of 19F and 31P NMR spectroscopy we studied these drugs in multicell tumor spheroids (MTS) formed by human colon carcinoma cells HT-29. This experimental system allowed detailed kinetic measurements of anabolic intracellular phosphates and fluorophosphates over periods of up to 2 days. Time-dependent NMR data were reduced and interpreted by the use of nonlinear compartmental models which yielded numerical values for the empirical rate constants characterizing mass transfer among the compartments. An analysis of these rate constants indicated qualitative and quantitative differences in the metabolism of FUra and FUrd and in the effects of GlcN on these drugs. The enhanced generation of FUDP-hexoses was a predicted effect of GlcN, but inhibited formation of fluorouridine diphosphates and fluorouridine diphosphates and fluorouridine triphosphates in FUra-treated MTS, and the magnitude of stimulation of fluoropyrimidine incorporation into macromolecules in FUrd-treated MTS were not predicted.
CT Medical Descriptors:
*colon cancer: DT, drug therapy
*tumor spheroid
*cancer chemotherapy
cytotoxicity
multicellular spheroid
nuclear magnetic resonance spectroscopy
phosphorus nuclear magnetic resonance
compartment model
macromolecule
drug metabolism

human
human cell
review
priority journal
Drug Descriptors:
*fluorouracil: IT, drug interaction
***fluorouracil: DT, drug therapy**
*fluorouracil: PK, pharmacokinetics
***fluorouridine: DT, drug therapy**
*fluorouridine: PK, pharmacokinetics
*glucosamine
fluoropyrimidine: IT, drug interaction
fluoropyrimidine: DT, drug therapy
fluoropyrimidine: PK, pharmacokinetics
phosphate: EC, endogenous compound
fluorophosphate: EC, endogenous compound
hexose
RN (fluorouracil) 51-21-8; (fluorouridine) 316-46-1; (glucosamine) **3416-24-8**, 4607-22-1; (fluoropyrimidine) 675-21-8; (phosphate) 14066-19-4, 14265-44-2; (fluorophosphate) 10163-15-2, 15181-43-8, 7631-97-2, 7789-74-4; (hexose) 93780-23-5

L122 ANSWER 17 OF 59 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1999:182014 BIOSIS
DOCUMENT NUMBER: PREV199900182014
TITLE: Concomitant cytokine delivery with poly-N-acetyl glucosamine (p-GlcNAc)/peptide vaccination leads to an enhanced CTL and anti-tumor response.
AUTHOR(S): Maitre, N.; Cole, D. J.; Stack, A.; Kelley, J. R.; Vary, C.; Demcheva, M.; Voumakis, J.
CORPORATE SOURCE: MUSC, Dep. Surgery, Hollings Cancer Cent., Charleston, SC USA
SOURCE: Proceedings of the American Association for Cancer Research Annual Meeting, (March, 1999) Vol. 40, pp. 79.
Meeting Info.: 90th Annual Meeting of the American Association for Cancer Research Philadelphia, Pennsylvania, USA April 10-14, 1999 American Association for Cancer Research
. ISSN: 0197-016X.
DOCUMENT TYPE: Conference
LANGUAGE: English
IT Major Concepts
Pharmacology; Tumor Biology
IT Parts, Structures, & Systems of Organisms
cytotoxic T-lymphocytes: blood and lymphatics, drug-induced activity enhancement, immune system
IT Diseases
cancer: immunochemotherapy, neoplastic disease
IT Chemicals & Biochemicals
granulocyte-macrophage colony stimulating factor: antineoplastic - drug, poly-N-acetylglucosamine-peptide vaccination delivery, immunologic - drug; interleukin-12: antineoplastic - drug, immunologic - drug, poly-N-acetylglucosamine-peptide vaccination delivery; interleukin-2: antineoplastic - drug, poly-N-acetylglucosamine-peptide vaccination delivery, immunologic - drug; transforming growth factor-beta soluble receptor II: antineoplastic - drug, immunologic - drug, poly-N-acetylglucosamine-peptide vaccination delivery
IT Alternate Indexing
Neoplasms (MeSH)
RN **3416-24-8** (GLUCOSAMINE)

L122 ANSWER 18 OF 59 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 1999005449 EMBASE
TITLE: A novel mutant from apoptosis-resistant colon cancer HT-29 cells showing hyper-apoptotic response to hypoxia, low glucose and cisplatin.
AUTHOR: Suzuki H.; Tomida A.; Tsuruo T.
CORPORATE SOURCE: T. Tsuruo, Laboratory of Biomedical Research, Inst Molecular Cellular Biosciences, University of Tokyo, 1-1-1 Yayoi, Bunkyo-ku, Tokyo 113-0032, Japan
SOURCE: Japanese Journal of Cancer Research, (1998) 89/11 (1169-1178).
Refs: 36
ISSN: 0910-5050 CODEN: JJCREP
COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Solid tumors usually have regions of hypoxia and glucose deprivation. Human colon carcinoma HT-29 cells show an apoptosis-resistant phenotype in response to microenvironmental stresses. In this study, we isolated a novel mutant of HT-29, designated as HA511, that showed a high apoptotic response to hypoxia, glucose deprivation and treatment with the chemical stressors tunicamycin and glucosamine. The mutant HA511 cells exhibited nuclear condensation and fragmentation and activation of CPP32 (caspase-3) protease under the stress conditions, while the parental HT-29 cells did not. We found that apoptosis occurred in HA511 cells after prolonged cell cycle arrest at the G1 phase, while in the parental cells a progression to S phase occurred after the G1 arrest. Upon exposure to an anti-Fas antibody, HA511 cells underwent apoptosis, whereas the parental cells proliferated without substantial cell death. Furthermore, HA511 cells were preferentially hypersensitive to cisplatin. We found no alteration in expression of GRP78, anti-apoptotic protein Bcl-X(L), or p53, of which the gene was mutated in HT-29 cells. The mutant HA511 cells could provide useful information on the mechanism of apoptosis of solid tumors.

CT Medical Descriptors:
*apoptosis
*colon cancer: DT, drug therapy
*colon cancer: ET, etiology
*hypoxia
phenotype
cell mutant
enzyme activation
cell cycle
cell cycle g1 phase
cell cycle s phase
cell death
human
controlled study
human cell
article
priority journal
Drug Descriptors:
*glucose
***cisplatin: DT, drug therapy**
tunicamycin
glucosamine
caspase 3: EC, endogenous compound

RN (glucose) 50-99-7, 84778-64-3; (cisplatin) 15663-27-1, 26035-31-4, 96081-74-2; (tunicamycin) 11089-65-9; (glucosamine) **3416-24-8**, 4607-22-1; (caspase 3) 169592-56-7

L122 ANSWER 19 OF 59 MEDLINE
ACCESSION NUMBER: 96064437 MEDLINE
DOCUMENT NUMBER: 96064437 PubMed ID: 8536261
TITLE: Versatile intermediates in the selective modification of the amino function of 2-amino-2-deoxy-D-mannopyranose and the 3-position of 2-acetamido-2-deoxy-D-mannose: potential membrane modifiers in neoplastic control.
AUTHOR: Angelino N J; Bernacki R J; Sharma M; Dodson-Simmons O; Korytnyk W
CORPORATE SOURCE: Department of Experimental Therapeutics, Grace Cancer Drug Center, Roswell Park Cancer Institute, Buffalo, NY 14263, USA.
CONTRACT NUMBER: CA13038 (NCI)
RO1 CA 08793 (NCI)
RO1 CA 42898 (NCI)
+
SOURCE: CARBOHYDRATE RESEARCH, (1995 Oct 16) 276 (1) 99-115.
Journal code: 0043535. ISSN: 0008-6215.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199602
ENTRY DATE: Entered STN: 19960221
Last Updated on STN: 19960221
Entered Medline: 19960205

AB A general method has been developed to selectively modify the amino group of 2-amino-2-deoxy-D-mannopyranose (D-mannosamine), a precursor of the terminal membrane sugar, sialic acid. 1,3,4,6-Tetra-O-acetyl-2-amino-2-deoxy-alpha-D-mannopyranose oxalate was prepared via two routes that allowed introduction of various acyl groups onto the amino function. These compounds were evaluated for their antineoplastic properties. The most significant preclinical therapeutic finding was the antileukemic activity found in mice for tetra-O-acetyl-2-epi-streptozotocin (the acetylated alpha-mannosamine epimer of streptozotocin). Administration of 50 mg/kg/day x 5 to leukemia L1210-bearing DBA/2Ha mice resulted in 5/5 35-day survivors. Neutralization of 1,3,4,6-tetra-O-acetyl-2-amino-2-deoxy-alpha-D-mannopyranose oxalate under aqueous conditions led to 2-acetamido-1,4,6-tri-O-acetyl-2-deoxy-alpha-D-mannopyranose, the oxidation of which gave 2-acetamido-4,6-di-O-acetyl-1,5-anhydro-2-deoxy-D-erythro-hex-1-en-3- ulose. This agent demonstrated an IC50(2) of 25 microM with a murine L1210 cell culture. Administration of 100 mg/kg/day x 5 resulted in 42% ILS3 in DBA/2 mice with ip L1210 leukemia. Several other nonacetylated derivatives were also prepared by direct N-acylation, producing, for example, fluorescently tagged N-dansylmannosamine.

CT Check Tags: Animal; Support, U.S. Gov't, P.H.S.
Antineoplastic Agents: CS, chemical synthesis
***Antineoplastic Agents: TU, therapeutic use**
Carbohydrate Conformation
Cell Membrane: DE, drug effects
*Cell Transformation, Neoplastic: DE, drug effects
Hexosamines: CS, chemical synthesis
*Hexosamines: CH, chemistry
*Hexosamines: TU, therapeutic use
*Leukemia L1210: DT, drug therapy
*Mannose: AA, analogs & derivatives

*Mannose: CH, chemistry
Mice
Mice, Inbred DBA
Sialic Acids: CH, chemistry
RN **2636-92-2 (mannosamine)**; 31103-86-3 (Mannose); 4773-29-9 (N-acetylmannosamine)

L122 ANSWER 20 OF 59 MEDLINE
ACCESSION NUMBER: 94179478 MEDLINE
DOCUMENT NUMBER: 94179478 PubMed ID: 8132768
TITLE: Marked elevation of plasma chitotriosidase activity. A novel hallmark of Gaucher disease.
AUTHOR: Hollak C E; van Weely S; van Oers M H; Aerts J M
CORPORATE SOURCE: Department of Biochemistry, Academic Medical Centre, Amsterdam, The Netherlands.
SOURCE: JOURNAL OF CLINICAL INVESTIGATION, (1994 Mar) 93 (3) 1288-92.
Journal code: 7802877. ISSN: 0021-9738.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199404
ENTRY DATE: Entered STN: 19940428
Last Updated on STN: 20000303
Entered Medline: 19940421

AB Gaucher disease (GD; glucosylceramidosis) is caused by a deficient activity of the enzyme glucocerebrosidase (GC). Clinical manifestations are highly variable and cannot be predicted accurately on the basis of the properties of mutant GC. Analysis of secondary abnormalities, such as elevated plasma levels of some hydrolases, may help to increase insight into the complicated pathophysiology of the disease and could also provide useful disease markers. The recent availability of enzyme supplementation therapy for GD increases the need for markers as early predictors of the efficacy of treatment. We report the finding of a very marked increase in chitotrisidase activity in plasma of 30 of 32 symptomatic type 1 GD patients studied: the median activity being > 600 times the median value in plasma of healthy volunteers. In three GC-deficient individuals without clinical symptoms, only slight increases were noted. Chitotriosidase activity was absent in plasma of three control subjects and two patients. During enzyme supplementation therapy, chitotriosidase activity declined dramatically. We conclude that plasma chitotriosidase levels can serve as a new diagnostic hallmark of GD and should prove to be useful in assessing whether clinical manifestations of GD are present and for monitoring the efficacy of therapeutic intervention.

CT Check Tags: Female; Human; Male
Adolescence
Adult
Aged
Alkaline Phosphatase: BL, blood
Child
Child, Preschool
Gaucher Disease: BL, blood
***Gaucher Disease: EN, enzymology**
*Hexosaminidases: BL, blood
Middle Age
Muramidase: BL, blood
*Trisaccharides: ME, metabolism
RN **41708-93-4 (chitotriose)**

L122 ANSWER 21 OF 59 MEDLINE
ACCESSION NUMBER: 95080876 MEDLINE
DOCUMENT NUMBER: 95080876 PubMed ID: 7989134
TITLE: Biological activities of chemically synthesized N-acylated serine-linked lipid A analog in mice.
AUTHOR: Shimizu T; Sugiyama K; Iwamoto Y; Yanagihara Y; Asahara T; Ikeda K; Achiwa K
CORPORATE SOURCE: Department of Microbiology, University of Shizuoka, School of Pharmaceutical Sciences, Japan.
SOURCE: INTERNATIONAL JOURNAL OF IMMUNOPHARMACOLOGY, (1994 Aug) 16 (8) 659-65.
Journal code: 7904799. ISSN: 0192-0561.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199501
ENTRY DATE: Entered STN: 19950124
Last Updated on STN: 19960129
Entered Medline: 19950110

AB The mitogenicity, lethal toxicity and antitumor activity against Meth A fibrosarcoma and the induction of tumor necrosis factor (TNF) of chemically synthesized N-acylated serine-linked nonphosphorylated acylglucosamine-derived lipid A analog (A-601, A-602 and A-603) were determined. Compounds A-603 (with (R)-3-tetradecanoyloxytetradecanoyl at the C-2 position) and A-103 (2,3-acyloxyacylglucosamine-4-phosphate) induced significant incorporations of [3H]thymidine into splenocytes of C3H/He mice at concentrations ranging from 6.25 to 100 microM. The mitogenicity of A-601 and A-602 (with tetradecanoyl at the C-2 position) exhibited a lower activity than of A-603. Compounds A-601 and A-603 showed almost the same lethality at doses from 1 to 50 nmol/mouse in C57BL/6 mice loaded with D-galactosamine, whereas A-103 caused the death of two out of six mice at a dose of 25 nmol/mouse. A-601 and A-603 showed weak antitumor activity against Meth A fibrosarcoma in BALB/c mice, but there was no enhancement of antitumor activity by a combination of A-603 with muramyl dipeptide. Peritoneal macrophages, stimulated with A-601, A-602 or A-603, caused production of TNF which induces L929 cell lysis in vitro. But the activity of A-603 among the compounds on TNF-production was the highest. These findings indicate that the linkage of nonphosphorylated acylglucosamine and N-acylated serine affects the expression of the biological activity.

CT Check Tags: Animal; Comparative Study
***Antineoplastic Agents: PD, pharmacology**
Antineoplastic Agents: TO, toxicity
Cell Division: DE, drug effects
Galactosamine: PD, pharmacology
Lethal Dose 50
*Lipid A: AA, analogs & derivatives
Lipid A: PD, pharmacology
Lipid A: TU, therapeutic use
Lipid A: TO, toxicity
Lipopolysaccharides: IP, isolation & purification
Lipopolysaccharides: PD, pharmacology
Mice
Mice, Inbred BALB C
Mice, Inbred C3H
Mice, Inbred C57BL
Salmonella typhimurium: CH, chemistry
Sarcoma, Experimental: DT, drug therapy
Tumor Necrosis Factor: BI, biosynthesis

RN 50696-27-0 (A 601); **7535-00-4 (Galactosamine)**

L122 ANSWER 22 OF 59 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 94076698 EMBASE
DOCUMENT NUMBER: 1994076698
TITLE: Detection of Tay-Sachs disease carriers among individuals with thermolabile hexosaminidase B.
AUTHOR: Peleg L.; Goldman B.
CORPORATE SOURCE: Genetic Institute, Sheba Medical Center,Tel-Hashomer 52621, Israel
SOURCE: European Journal of Clinical Chemistry and Clinical Biochemistry, (1994) 32/2 (65-69).
ISSN: 0939-4974 CODEN: EJCBEO
COUNTRY: Germany
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 008 Neurology and Neurosurgery
012 Ophthalmology
022 Human Genetics
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

AB The determination of hexosaminidases A and B in most programmes for Tay-Sachs disease carrier detection is based on their different heat sensitivity (hexosaminidase A is the heat labile isoenzyme). This routine cannot be employed for individuals who also possess a thermolabile hexosaminidase B. In Israel, 0.6% of the screened samples have a labile hexosaminidases B (about 110 each year) and the assessment of their hexosaminidase A activity has hitherto been based on isoenzyme separation by ion exchange chromatography. The latter requires relative large serum samples, and the individuals must usually be reappointed. In order to avoid the thermal treatment we have used the alternative technique, which employs two substrates with different specificities for the two isoenzymes: 1. The fluorogenic substance, 4-methylumbelliferyl-N-acetyl-glucopyranoside, which measures total hexosaminidase activity and 2. the derivative, 4-methylumbelliferyl-N-acetyl glucosamine-6-sulphate, which is considerably more specific toward hexosaminidase A. Hexosaminidase A activity was expressed as a ratio of total activities (the ratio of the assay with 4-methylumbelliferyl-N-acetyl glucosamine-6-sulphate to that with 4-methylumbelliferyl-N-acetyl-glucopyranoside). Using the results from 65 obligate heterozygotes for Tay-Sachs disease, we established our reference ranges for assigning the genotypes with respect to the Tay-Sachs gene. Comparison of the results from 182 unrelated and randomly chosen sera screened by the ratio method and by heat inactivation, showed a very high correlation (r = 0.996). Sixty eight sera with thermolabile hexosaminidase B were tested by ion exchange chromatography and by the double substrate method, and they yielded identical diagnoses with regard to the Tay-Sachs locus. The latter strategy showed an improved inter-assay coefficient of variation (11% instead of 21%); it also utilizes very small amounts of sera. Results for the estimation of hexosaminidase B heat sensitivity are also presented and analysed.

CT Medical Descriptors:
*heterozygote detection
***tay sachs disease: CN, congenital disorder**
article
comparative study
controlled study
enzyme activity
enzyme isolation
enzyme specificity
gene locus

genotype
heat sensitivity
heat treatment
human
human cell
ion exchange chromatography
israel
normal human
priority journal
statistical analysis
Drug Descriptors:
*beta n acetylhexosaminidase b: EC, endogenous compound
beta n acetylhexosaminidase a: EC, endogenous compound
glucosamine derivative
glucosamine sulfate
isoenzyme
RN (glucosamine sulfate) **29031-19-4**

L122 ANSWER 23 OF 59 MEDLINE
ACCESSION NUMBER: 93119528 MEDLINE
DOCUMENT NUMBER: 93119528 PubMed ID: 1476672
TITLE: Relation between the biologic activities and chemical structures of synthetic microbial lipopeptide analogs in mice.
AUTHOR: Shimizu T; Haketa Y; Iwamoto Y; Yanagihara Y; Kurimura M; Ochiai A; Achiwa K
CORPORATE SOURCE: Department of Microbiology, University of Shizuoka, School of Pharmaceutical Sciences, Japan.
SOURCE: MOLECULAR BIOTHERAPY, (1992 Dec) 4 (4) 184-7.
Journal code: 8904897. ISSN: 0952-8172.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199302
ENTRY DATE: Entered STN: 19930226
Last Updated on STN: 19930226
Entered Medline: 19930210

AB Mitogenicity, lethal toxicity, and antitumor activity against Meth A fibrosarcoma of chemically synthesized lipopeptide analogs, S-[2,3-bis(palmitoyloxy)-2R-propyl]-N-[(2,2,2)-tri- chloroethoxycarbonyl: Troc group]-cysteinyl-seryl-seryl-asparaginyl-alanine (compound KAB-2), which contain the amino acid sequence of lipopeptide in Escherichia coli, S-[2,3-bis(palmitoyloxy)- 2R-propyl]-N-(Troc- or amino-group)-cysteinyl-asparaginyl-seryl-glycyl-glycine (compound KAB-14 or -20), which is found in the amino acid sequence of lipopeptide in Streptomyces, and the compounds binding one to six amino acids, were examined. The analogs showed the mitogenic activity toward splenocytes of C3H/He mice. Low concentrations (0.4 and 2.0 micrograms/ml) of compounds KAB-20 and -21, which have five and six amino acids, respectively, increased the incorporation of [3H]thymidine better than a high concentration (50 micrograms/ml), suggesting that KAB compounds carrying amino groups exert better mitogenicity than KAB compounds carrying Troc group. The decrease of amino acid number in lipopeptide analogs appears to result in a lowering of mitogenicity at low concentrations. KAB-14 and KAB-2 did not exhibit the lethality at a high dose of 50 micrograms/mouse in galactosamine-loaded C57BL/6 mice. By twice intravenous injections of 50 micrograms against Meth A fibrosarcoma in BALB/c mice, KAB-2 showed a higher inhibitory effect than KAB-14. Based on these results, we concluded that the difference of amino acid sequence in the synthetic lipopeptides

affects the potency of biologic activities.
CT Check Tags: Animal; Male
Amino Acid Sequence
Antineoplastic Agents: CS, chemical synthesis
***Antineoplastic Agents: PD, pharmacology**
Antineoplastic Agents: TO, toxicity
Drug Screening Assays, Antitumor
*Fibrosarcoma: DT, drug therapy
Galactosamine: PD, pharmacology
Lipoproteins: CS, chemical synthesis
*Lipoproteins: PD, pharmacology
Lipoproteins: TO, toxicity
Mice
Mice, Inbred BALB C
Mice, Inbred C3H
Mice, Inbred C57BL
Molecular Sequence Data
RN **7535-00-4 (Galactosamine)**

L122 ANSWER 24 OF 59 MEDLINE
ACCESSION NUMBER: 92182253 MEDLINE
DOCUMENT NUMBER: 92182253 PubMed ID: 1665717
TITLE: Receptor-mediated protection of normal hepatocytes during chemotherapy for hepatocellular carcinoma.
AUTHOR: Keegan-Rogers V; Wu C H; Wu G Y
CORPORATE SOURCE: University of Connecticut School of Medicine, Farmington.
CONTRACT NUMBER: CA 01110 (NCI)
CA 46801 (NCI)
SOURCE: TARGETED DIAGNOSIS AND THERAPY, (1991) 4 105-25. Ref: 71
Journal code: 8913519. ISSN: 1046-1906.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199204
ENTRY DATE: Entered STN: 19920424
Last Updated on STN: 19920424
Entered Medline: 19920413
CT Check Tags: Animal; Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
Antineoplastic Agents: AD, administration & dosage
***Antineoplastic Agents: TU, therapeutic use**
*Carcinoma, Hepatocellular: DT, drug therapy
Cells, Cultured
Galactosamine: TO, toxicity
Liver: CY, cytology
Liver: DE, drug effects
*Liver: PH, physiology
*Liver Neoplasms: DT, drug therapy
*Liver Neoplasms, Experimental: DT, drug therapy
Receptors, Immunologic: DE, drug effects
*Receptors, Immunologic: PH, physiology
RN **7535-00-4 (Galactosamine)**

L122 ANSWER 25 OF 59 MEDLINE
ACCESSION NUMBER: 91162596 MEDLINE
DOCUMENT NUMBER: 91162596 PubMed ID: 1825851
TITLE: Tay-Sachs disease heterozygote detection: use of a

centrifugal analyser for automation of hexosaminidase assays with two different artificial substrates.
AUTHOR: Landels E C; Ellis I H; Bobrow M; Fensom A H
CORPORATE SOURCE: Division of Medical and Molecular Genetics, United Medical School of Guy's Hospital, London.
SOURCE: JOURNAL OF MEDICAL GENETICS, (1991 Feb) 28 (2) 101-9. Journal code: 2985087R. ISSN: 0022-2593.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199104
ENTRY DATE: Entered STN: 19910505
Last Updated on STN: 19910505
Entered Medline: 19910412

AB An assay for measuring hexosaminidase A in serum and leucocytes is described in which a centrifugal analyser is used for automation of the enzyme assays after manual heat inactivation. The assay was used in a screening programme to identify heterozygotes for Tay-Sachs disease in Ashkenazi Jewish subjects in the UK. The first results from this programme indicate a carrier frequency of 1 in 27. Automation of an assay for direct measurement of hexosaminidase A in serum using 4-methyl-umbelliferyl-beta-N-acetylglucosamine-6-sulphate as substrate is also described. Comparison of data obtained from 66 control and 30 obligate carrier sera tested by this method and by heat inactivation showed improved discrimination using the sulphated substrate. Results obtained using the sulphated substrate for screening serum during pregnancy are also presented.

CT Check Tags: Comparative Study; Female; Human; Support, Non-U.S. Gov't
Automation
Cell Separation
Centrifugation
Flow Cytometry
*Genetic Screening
Glucosamine: AA, analogs & derivatives
Glucosamine: DU, diagnostic use
Heat
*Heterozygote Detection: MT, methods
Hymecromone: AA, analogs & derivatives
Hymecromone: DU, diagnostic use
Leukocytes: EN, enzymology
Pregnancy
Prenatal Diagnosis
***Tay-Sachs Disease: DI, diagnosis**
Tay-Sachs Disease: EN, enzymology
Tay-Sachs Disease: GE, genetics
beta-N-Acetylhexosaminidase: AN, analysis
*beta-N-Acetylhexosaminidase: BL, blood

RN **3416-24-8 (Glucosamine)**; 37067-30-4 (4-methylumbelliferyl 2-acetamido-2-deoxy-beta-D-glucopyranoside); 90-33-5 (Hymecromone); 93751-71-4 (4-methylumbelliferyl-6-sulfo-2-acetamido-2-deoxy-beta-glucopyranoside)

L122 ANSWER 26 OF 59 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 89095964 EMBASE
DOCUMENT NUMBER: 1989095964
TITLE: Treatment of chronic liver injury in mice by oral administration of Xiao-Chai-Hu-Tang.
AUTHOR: Amagaya S.; Hayakawa M.; Ogihara Y.; Ohta Y.; Fujiwara K.; Oka H.; Oshio H.; Kishi T.
CORPORATE SOURCE: Faculty of Pharmaceutical Science, Nagoya City University,

Nagoya 467, Japan
SOURCE: Journal of Ethnopharmacology, (1989) 25/2 (181-187).
ISSN: 0378-8741 CODEN: JOETD7
COUNTRY: Ireland
DOCUMENT TYPE: Journal
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
048 Gastroenterology
052 Toxicology
037 Drug Literature Index
LANGUAGE: English
CT Medical Descriptors:
*liver injury: DT, drug therapy
*liver toxicity
animal model
histology
mouse
prothrombin time
animal experiment
nonhuman
plant
male
oral drug administration
Drug Descriptors:
alanine aminotransferase
carbon tetrachloride
galactosamine
hydroxyproline
***xiao chai hu tang: DT, drug therapy**
RN (alanine aminotransferase) 9000-86-6, 9014-30-6; (carbon tetrachloride) 56-23-5; (galactosamine) **7535-00-4**; (hydroxyproline) 51-35-4, 6912-67-0; (xiao chai hu tang) 63364-01-2

L122 ANSWER 27 OF 59 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 89124915 EMBASE
DOCUMENT NUMBER: 1989124915
TITLE: Antimetabolites.
AUTHOR: Allegra C.J.; Grem J.L.; Yeh G.C.; Chabner B.A.
CORPORATE SOURCE: Clinical Pharmacology Branch, Division of Cancer Treatment, National Cancer Institute, National Institutes of Health, Bethesda, MD 20892, United States
SOURCE: Cancer chemotherapy and biological response modifiers. Annual 10, (1988) (1-22).Editor: Pinedo H.M.; Longo D.L.; Chabner B.A. Publisher: Elsevier Science Publishers B.V.
ISBN: 0444810153
DOCUMENT TYPE: Book; Journal
FILE SEGMENT: 016 Cancer
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The mechanisms of action of MTX and 5-FU have been further elucidated. Such studies will be important for the design of drug combinations and for the development of novel antifolate and fluoropyrimidine analogs. A greater understanding of MTX and ara-C transport and drug levels required to optimize transport may also aid in these endeavors. Pharmacokinetic parameters have been found to be predictors of relapse in children with acute leukemia, particularly with respect to MTX, 6-MP and ara-C. The intracellular terminal half-life of ara-C was correlated with remission duration in AML. Assay systems aimed at uncovering response predictors through biochemical analysis of patient tumor samples are being developed,

including an interesting use of NMR spectroscopy to study the pharmacokinetics of fluorine-19-labeled 5-FU in vivo. Such an approach may yield valuable information on 5-FU anabolism in tumors in situ. A high frequency of resistance to MTX apparently may be generated within a single cell cycle by transient exposures to DNA synthesis inhibitors. The resistance may be based on either target enzyme amplification or altered membrane transport. These important studies provided bases for the rapid emergence of clinical resistance. Further, the multidrug-resistant phenotype appears to be a much broader based phenomenon as MTX resistance was found to be a frequent event in cells selected for multidrug resistance. A variety of novel approaches have been proposed to overcome antimetabolite resistance and to improve the selectivity of these agents, including the use of guanosine nucleotides, leucovorin and allopurines as biochemical modulators of 5-FU. Efficient techniques for the transfection of resistant DHFR into tissues using retroviruses have been reported. These studies serve as starting point for the ultimate development of more effective strategies for the treatment of human malignancies.

CT Medical Descriptors:
*cancer: DT, drug therapy
*drug mechanism
*drug metabolism
*drug resistance
*drug transport
*pharmacokinetics
aseptic meningitis: SI, side effect
bone marrow suppression: SI, side effect
lung infiltrate: SI, side effect
review
human
nonhuman
Drug Descriptors:
*allopurinol: CB, drug combination
*allopurinol: IT, drug interaction
*cisplatin: CB, drug combination
*cisplatin: IT, drug interaction
*cytarabine: PK, pharmacokinetics
*cytarabine: TO, drug toxicity
***cytarabine: DT, drug therapy**
*cytarabine: PD, pharmacology
*cytarabine: AE, adverse drug reaction
*fluorouracil: PK, pharmacokinetics
*fluorouracil: TO, drug toxicity
*fluorouracil: AE, adverse drug reaction
*fluorouracil: PD, pharmacology
*fluorouracil: IT, drug interaction
***fluorouracil: DT, drug therapy**
*fluorouracil: CB, drug combination
*folinic acid: IT, drug interaction
*folinic acid: CB, drug combination
*glucosamine: CB, drug combination
*glucosamine: IT, drug interaction
*granulocyte colony stimulating factor: CB, drug combination
*granulocyte colony stimulating factor: IT, drug interaction
*guanosine diphosphate: CB, drug combination
*guanosine diphosphate: IT, drug interaction
*guanosine phosphate: CB, drug combination
*guanosine phosphate: IT, drug interaction
*guanosine triphosphate: IT, drug interaction
*guanosine triphosphate: CB, drug combination
*histidinol: IT, drug interaction

*histidinol: CB, drug combination
*idoxuridine: CB, drug combination
*idoxuridine: IT, drug interaction
*mercaptopurine: PD, pharmacology
*mercaptopurine: PK, pharmacokinetics
***mercaptopurine: DT, drug therapy**
***methotrexate: DT, drug therapy**
*methotrexate: PK, pharmacokinetics
*methotrexate: CB, drug combination
*methotrexate: PD, pharmacology
***methotrexate derivative: DT, drug therapy**
*methotrexate derivative: IT, drug interaction
*methotrexate derivative: PD, pharmacology
*pentostatin: PD, pharmacology
*pentostatin: PK, pharmacokinetics
***pentostatin: DT, drug therapy**
*sparfosic acid: CB, drug combination
*sparfosic acid: IT, drug interaction
*tioguanine: PK, pharmacokinetics
***tioguanine: DT, drug therapy**
*tioguanine: PD, pharmacology
*vidarabine derivative: PK, pharmacokinetics
*vidarabine derivative: DT, drug therapy
*vidarabine derivative: PD, pharmacology

RN (allopurinol) 315-30-0; (cisplatin) 15663-27-1, 26035-31-4, 96081-74-2; (cytarabine) 147-94-4, 69-74-9; (fluorouracil) 51-21-8; (folinic acid) 58-05-9, 68538-85-2; (glucosamine) **3416-24-8**, 4607-22-1; (guanosine diphosphate) 146-91-8; (guanosine phosphate) 29593-02-0, 5550-12-9, 85-32-5; (guanosine triphosphate) 86-01-1; (histidinol) 501-28-0; (idoxuridine) 54-42-2; (mercaptopurine) 31441-78-8, 50-44-2, 6112-76-1; (methotrexate) 15475-56-6, 59-05-2, 7413-34-5; (pentostatin) 53910-25-1; (sparfosic acid) 51321-79-0; (tioguanine) 154-42-7

L122 ANSWER 28 OF 59 MEDLINE
ACCESSION NUMBER: 88258842 MEDLINE
DOCUMENT NUMBER: 88258842 PubMed ID: 3385605
TITLE: Antitumor activity of polygalactosamine isolated from Paecilomyces sp. I-1 strain.
AUTHOR: Ishitani K; Suzuki S; Suzuki M
CORPORATE SOURCE: Department of Microbiology, Tohoku College of Pharmacy, Sendai, Japan.
SOURCE: JOURNAL OF PHARMACOBIO-DYNAMICS, (1988 Jan) 11 (1) 58-65. Journal code: 7901854. ISSN: 0386-846X.
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198808
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 19990129
Entered Medline: 19880801

AB The inhibitory effect of polygalactosamine (PF102), which was isolated from Paecilomyces sp. I-1 strain, on a syngeneic murine solid tumor and its antitumor mechanism were studied. After an intravenous injection of PF102, 1 microgram/kg, an increase in cell mediated and humoral immunities in mice was observed and the growth inhibition of MM46 solid tumor in vivo was also evident. Macrophages induced by PF102 into the peritoneal cavity inhibited deoxyribonucleic acid synthesis of target cells. Moreover, PF102 caused a significant increase in the incorporation of 3H-thymidine into the thymic cells and the culture supernatant of T lymphocytes, stimulated

with PF102, exhibited a marked activation of the cytostatic effect of the peritoneal macrophages. Furthermore, this culture supernatant fluid was found to contain interferon (IFN). Therefore, the antitumor activity of PF102 might be due in part to the activation of the macrophage lineage cells by macrophage activating factor and/or IFN produced from T lymphocytes stimulated by PF102.

CT Check Tags: Animal; Male
Antibody Formation
Antineoplastic Agents: IP, isolation & purification
***Antineoplastic Agents: PD, pharmacology**
Cell Division
Galactosamine: IP, isolation & purification
*Galactosamine: PD, pharmacology
*Hypersensitivity, Delayed
Immunity, Cellular
Mice
Mice, Inbred C3H
*Mitosporic Fungi: AN, analysis
*Paecilomyces: AN, analysis
*Tumor Cells, Cultured: DE, drug effects
Tumor Cells, Cultured: IM, immunology

RN **7535-00-4 (Galactosamine)**

L122 ANSWER 29 OF 59 MEDLINE
ACCESSION NUMBER: 87216518 MEDLINE
DOCUMENT NUMBER: 87216518 PubMed ID: 3107855
TITLE: Effects of exogenous beta-galactosidase on cultured fibroblasts with beta-galactosidase deficiency.
AUTHOR: Nakao Y; Kozutsumi Y; Fukui S; Kawasaki T; Yamashina I
SOURCE: CLINICA CHIMICA ACTA, (1987 Apr 15) 164 (1) 101-7. Journal code: 1302422. ISSN: 0009-8981.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198706
ENTRY DATE: Entered STN: 19900303
Last Updated on STN: 19900303
Entered Medline: 19870626

CT Check Tags: Human; Support, Non-U.S. Gov't
*Carbohydrates: ME, metabolism
Cells, Cultured
Chromatography, Gel
Fibroblasts: ME, metabolism
*Galactosidases: ME, metabolism
***Gangliosidoses: ME, metabolism**
Glucosamine: ME, metabolism
Liposomes: ME, metabolism
beta-Galactosidase: DF, deficiency
*beta-Galactosidase: ME, metabolism

RN **3416-24-8 (Glucosamine)**

L122 ANSWER 30 OF 59 MEDLINE
ACCESSION NUMBER: 87299749 MEDLINE
DOCUMENT NUMBER: 87299749 PubMed ID: 2956992
TITLE: Human acid beta-glucosidase: use of inhibitors, alternative substrates and amphiphiles to investigate the properties of the normal and Gaucher disease active sites.
AUTHOR: Osiecki-Newman K; Fabbro D; Legler G; Desnick R J; Grabowski G A

CONTRACT NUMBER: K04 AM01351 (NIADDK)
R01 AM 26729 (NIADDK)
RR-71 (NCRR)
SOURCE: BIOCHIMICA ET BIOPHYSICA ACTA, (1987 Sep 2) 915 (1) 87-100.
Journal code: 0217513. ISSN: 0006-3002.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198710
ENTRY DATE: Entered STN: 19900305
Last Updated on STN: 20000303
Entered Medline: 19871013

AB Comparative studies with lipoidal inhibitors and alternative substrates were conducted to investigate the properties of the active site of human acid beta-glucosidase (D-glucosyl-N-acylsphingosine glucohydrolase, EC 3.2.1.45) from normal placenta and spleens of Type 1 Ashkenazi Jewish Gaucher disease (AJGD) patients. With the normal enzyme, the inhibitory potencies of series of alkyl(Cn; n = 0-18)amines, alkyl beta-glucosides and alkyl-1-deoxynojirimycins were a biphasic function of increasing chain length: i.e., large decreases in Ki,app or IC50 were found only with n greater than 4 and limiting values were approached with n = 12-14. This biphasic function of alkyl chain length was observed in the presence or absence of detergents and/or negatively charged lipids. In the presence of Triton X-100 concentrations greater than the critical micellar concentration, the relative (to deoxynojirimycin) inhibitory potencies of the N-Cn-deoxynojirimycins (n greater than 4) were decreased about 3-5-fold, due to an energy requirement to extract the inhibitors from Triton X-100 micelles. The Ki,app or IC50 of N-hexylglucosylsphingosine was inversely related to the Triton X-100 concentration and was not affected by the presence of 'co-glucosidase'. The mutual exclusion of glucon, N-Cn-deoxynojirimycin and sphingosine derivatives from the normal enzyme suggested a shared region for binding in the active site. Increasing the fatty-acid acyl chain length of glucosyl ceramide from 1 to 24 carbons had minor effects on Km,app (= Kis,app) (8-40 microM), but increased Vmax,app up to 13-fold. With the AJGD enzyme, the inhibitor and alternative substrate findings were similar to those with the normal enzyme, except that Kis,app(AJGD)/Kis,app(normal) = 4 to 11 for the Cn-glycons and sphingosine derivatives. These results indicated that (1) the Ki,app or Km,app values for amphiphilic inhibitors or substrates reflect a balance of binding energies for two hydrophobic subsites within the enzyme's active site and Triton X-100 micelles and (2) the abnormal properties of the AJGD enzyme result from an amino-acid alteration(s) within or near a hydrophilic region which is shared by the glycon-binding site and the two hydrophobic sites of the active site.

CT Check Tags: Female; Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.

1-Deoxynojirimycin
Amines: PD, pharmacology
Binding Sites
Binding, Competitive
Ceramides: ME, metabolism
***Gaucher Disease: EN, enzymology**
Glucosamine: AA, analogs & derivatives
Glucosamine: PD, pharmacology
*Glucosidases: ME, metabolism
Glucosides: PD, pharmacology
Kinetics
Octoxynol
Placenta: EN, enzymology

Polyethylene Glycols: PD, pharmacology
Pregnancy
Sphingosine: AA, analogs & derivatives
Sphingosine: PD, pharmacology
Spleen: EN, enzymology
Structure-Activity Relationship
beta-Glucosidase: AI, antagonists & inhibitors
*beta-Glucosidase: ME, metabolism
RN 123-78-4 (Sphingosine); 19130-96-2 (1-Deoxynojirimycin); **3416-24-8 (Glucosamine)**; 9002-93-1 (Octoxynol)

L122 ANSWER 31 OF 59 MEDLINE
ACCESSION NUMBER: 86172726 MEDLINE
DOCUMENT NUMBER: 86172726 PubMed ID: 3457206
TITLE: Interference with tumor cell-induced degradation of endothelial matrix on the antimetastatic action of nafazatrom.
AUTHOR: Maniglia C A; Loulakis P P; Sartorelli A C
CONTRACT NUMBER: CA-02817 (NCI)
SOURCE: JOURNAL OF THE NATIONAL CANCER INSTITUTE, (1986 Apr) 76 (4) 739-44.
Journal code: 7503089. ISSN: 0027-8874.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198605
ENTRY DATE: Entered STN: 19900321
Last Updated on STN: 19970203
Entered Medline: 19860505

AB The antithrombotic compound nafazatrom was evaluated in several in vivo and in vitro assays to elucidate the mechanism of its antimetastatic activity. C57BL/6 mice bearing B16 amelanotic subcutaneous tumors treated with 100 mg nafazatrom/kg/day exhibited a sixfold reduction in metastatic pulmonary lesions compared to lesion numbers in controls. The reduction in metastatic lesions was not accompanied by changes in primary tumor growth, and up to 1 microgram nafazatrom/ml did not inhibit tumor cell proliferation in vitro. Treatment of C57BL/6 mice with nafazatrom prior to iv inoculation of tumor cells failed to inhibit lung colony formation. In vitro exposure of exponentially growing B16 amelanotic cells to nafazatrom (1 microgram/ml for 72 hr) in culture did not change their ability to adhere to endothelial cell monolayers. B16 amelanotic cells degraded the matrix material of bovine endothelial cell monolayers; a heparin sulfate proteoglycan appeared to be the predominant matrix component released by these tumor cells, as judged by resistance to chondroitin ABC lyase and sensitivity to heparitinase and pronase degradation. Nafazatrom (1 microgram/ml for 72 hr) inhibited the solubilization of matrix components by approximately 60%. Tumor cell degradation of matrix components is an important event in the pathogenesis of metastasis. Thus the interference with this process appears to provide an explanation for the inhibition of malignant cell dissemination in vivo by nafazatrom.

CT Check Tags: Animal; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
***Antineoplastic Agents: PD, pharmacology**
*Blood Vessels: ME, metabolism
Cell Adhesion
Cell Division: DE, drug effects
Cells, Cultured
Endothelium: ME, metabolism
*Extracellular Matrix: ME, metabolism
Glucosamine: ME, metabolism

Melanoma: ME, metabolism
Melanoma: PA, pathology
Mice
Mice, Inbred C57BL
*Neoplasm Metastasis
*Pyrazoles: PD, pharmacology
RN **3416-24-8 (Glucosamine)**; 59040-30-1 (nafazatrom)

L122 ANSWER 32 OF 59 MEDLINE
ACCESSION NUMBER: 87117959 MEDLINE
DOCUMENT NUMBER: 87117959 PubMed ID: 3809110
TITLE: Prenatal diagnosis of infantile GM 2 gangliosidosis type II (Sandhoff disease) by detection of N-acetylglucosaminyl-oligosaccharides in amniotic fluid with high-performance liquid chromatography.
AUTHOR: Warner T G; Turner M W; Toone J R; Applegarth D
CONTRACT NUMBER: NS 22323 (NINDS)
SOURCE: PRENATAL DIAGNOSIS, (1986 Nov-Dec) 6 (6) 393-400. Journal code: 8106540. ISSN: 0197-3851.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198703
ENTRY DATE: Entered STN: 19900303
Last Updated on STN: 19970203
Entered Medline: 19870304

AB Prenatal diagnosis of Sandhoff disease (infantile onset) at 16 weeks gestation has been made by detection and analysis of N-acetylglucosaminyl-oligosaccharides in amniotic fluid using high performance liquid chromatography. The elution profile for the branched chain oligosaccharides was identical with that obtained with neonatal and infantile Sandhoff urine. The concentration of the oligosaccharides in the fluid was 1/100th that of urine but when calculated relative to creatinine the levels were similar. No oligosaccharides were detected in normal control amniotic fluids (10 patients) at a similar gestational age. Based on the levels of the amniotic fluid oligosaccharides and the sensitivity limits of the assay, prenatal diagnosis of patients with the juvenile onset form of the disease may also be possible with this technique.

CT Check Tags: Female; Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
*Acetylglucosamine: AN, analysis
Acetylglucosamine: UR, urine
*Amniotic Fluid: AN, analysis
Chromatography, High Pressure Liquid
*Glucosamine: AA, analogs & derivatives
*Oligosaccharides: AN, analysis
Oligosaccharides: UR, urine
Pregnancy
*Prenatal Diagnosis
***Sandhoff Disease: DI, diagnosis**
RN **3416-24-8 (Glucosamine)**; 7512-17-6 (Acetylglucosamine)

L122 ANSWER 33 OF 59 MEDLINE
ACCESSION NUMBER: 87004496 MEDLINE
DOCUMENT NUMBER: 87004496 PubMed ID: 2944742
TITLE: Human acid beta-glucosidase: affinity purification of the normal placental and Gaucher disease splenic enzymes on N-alkyl-deoxynojirimycin-sepharose.
AUTHOR: Osiecki-Newman K M; Fabbro D; Dinur T; Boas S; Gatt S;

Legler G; Desnick R J; Grabowski G A
CONTRACT NUMBER: AM 36729 (NIADDK)
K04-AM 01351 (NIADDK)
SOURCE: ENZYME, (1986) 35 (3) 147-53.
Journal code: 1262265. ISSN: 0013-9432.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198610
ENTRY DATE: Entered STN: 19900302
Last Updated on STN: 20000303
Entered Medline: 19861030

AB Two sepharose-bound 1-deoxynojirimycin N-alkyl derivatives, N-(9-carboxynonyl)- and N-(11-carboxyundecyl)-deoxynojirimycin, were used for the affinity purification of acid beta-glucosidase (beta-Glc) from normal and type-1 Ashkenazi Jewish Gaucher disease (AJGD) sources. The capacities of these nondegradable inhibitor supports were 0.5 and 0.75 mg of normal beta-Glc/ml of settled gel, respectively. The purified normal enzyme (14-18% yield) had a specific activity of 1.6 X 10(6) nmol/h/mg protein and was homogeneous as evidenced by a single protein species of Mr = 67,000 on sodium dodecylsulfate-polyacrylamide gel electrophoresis and reverse phase high-performance liquid chromatography (HPLC). Microsequencing demonstrated a single N terminus, and the sequence of the first 22 N-terminal amino acids was colinear with that predicted from the beta-Glc cDNA. Amino acid composition analyses of beta-Glc revealed a high content (35%) of hydrophobic amino acids. The N-decyl-deoxynojirimycin support facilitated the purification of the residual enzyme from type-1 AJGD spleen to about 7,500-fold in four steps with a yield of about 11%. These new affinity supports provided improved stability, capacity and/or specificity compared to other affinity or HPLC methods for purifying this lysosomal glycosidase.

CT Check Tags: Comparative Study; Female; Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
1-Deoxynojirimycin
Amino Acid Sequence
Chromatography, Affinity
Chromatography, High Pressure Liquid
Electrophoresis, Polyacrylamide Gel
***Gaucher Disease: EN, enzymology**
Glucosamine: AA, analogs & derivatives
*Glucosidases: IP, isolation & purification
Peptide Fragments
*Placenta: EN, enzymology
Pregnancy
*Spleen: EN, enzymology
*beta-Glucosidase: IP, isolation & purification

RN 19130-96-2 (1-Deoxynojirimycin); **3416-24-8 (Glucosamine)**

L122 ANSWER 34 OF 59 MEDLINE
ACCESSION NUMBER: 86195136 MEDLINE
DOCUMENT NUMBER: 86195136 PubMed ID: 2422137
TITLE: 3-Deazauridine (NSC 126849): an interesting modulator of biochemical response.
AUTHOR: Moriconi W J; Slavik M; Taylor S
SOURCE: INVESTIGATIONAL NEW DRUGS, (1986) 4 (1) 67-84.
Journal code: 8309330. ISSN: 0167-6997.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English

FILE SEGMENT: Priority Journals
ENTRY MONTH: 198606
ENTRY DATE: Entered STN: 19900321
Last Updated on STN: 19970203
Entered Medline: 19860606

AB 3-Deazauridine (NSC 126849) is a structural analog of uridine that inhibits the biosynthesis of Cytidine-5'-Triphosphate by competitive inhibition of Cytidine Triphosphate synthetase which is considered to be the primary mode of action of this nucleoside analog. Despite a paucity of clinical attention given to this drug as a single agent, it has generated much enthusiasm as a biological response modulator because of its synergistic effect with a number of antitumor agents including Cytosine Arabinoside, 5-aza-2'-deoxycytidine, 5-azacytidine, thymidine and D-galactosamine, although only the cytosine arabinoside/3-Deazauridine combination has been explored clinically. In this paper, the current status of the drug and future perspectives will be discussed.

CT Check Tags: Animal; Human
3-Deazauridine: ME, metabolism
*3-Deazauridine: PD, pharmacology
3-Deazauridine: TU, therapeutic use
3-Deazauridine: TO, toxicity
Acute Disease
Antineoplastic Agents: ME, metabolism
***Antineoplastic Agents: PD, pharmacology**
Antineoplastic Agents: TU, therapeutic use
Antineoplastic Agents: TO, toxicity
Azacitidine: AA, analogs & derivatives
Azacitidine: PD, pharmacology
Cytarabine: PD, pharmacology
Drug Evaluation
Drug Synergism
Galactosamine: PD, pharmacology
Kinetics
Leukemia: DT, drug therapy
Neoplasms: DT, drug therapy
Thymidine: PD, pharmacology
*Uridine: AA, analogs & derivatives

RN 147-94-4 (Cytarabine); 23205-42-7 (3-Deazauridine); 2353-33-5 (5-aza-2'-deoxycytidine); 320-67-2 (Azacitidine); 50-89-5 (Thymidine); 58-96-8 (Uridine); **7535-00-4 (Galactosamine)**

L122 ANSWER 35 OF 59 MEDLINE
ACCESSION NUMBER: 85207603 MEDLINE
DOCUMENT NUMBER: 85207603 PubMed ID: 3997819
TITLE: Characterization and analysis of branched-chain N-acetylglucosaminyl oligosaccharides accumulating in Sandhoff disease tissue. Evidence that biantennary bisected oligosaccharide side chains of glycoproteins are abundant substrates for lysosomes.
AUTHOR: Warner T G; deKremer R D; Sjoberg E R; Mock A K
CONTRACT NUMBER: NS 2232 (NINDS)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1985 May 25) 260 (10) 6194-9.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198506
ENTRY DATE: Entered STN: 19900320

Last Updated on STN: 19970203
Entered Medline: 19850627

AB Branched chain N-acetylglucosaminyl oligosaccharides accumulating in visceral and neural tissues of two patients with Sandhoff disease were isolated and quantified using high performance liquid chromatography. Detailed structural analysis of the three most abundant fractions, oligosaccharides 4, 5, and 6, was carried out using 360 MHz proton magnetic resonance spectroscopy. The biantennary bisected heptasaccharide, oligosaccharide 6, was ubiquitously distributed and a major component of the stored oligosaccharides in all tissues analyzed including, liver, spleen, kidney, lung, pancreas, and brain. This analysis indicates that glycoproteins containing biantennary bisected oligosaccharide side chains are abundant substrates for lysosomes in human tissues. Moreover, oligosaccharide 6 was the predominant storage product in brain comprising 70% of the total accumulating water-soluble glycoconjugates. Oligosaccharide 5, a triantennary heptasaccharide, had a similar distribution in visceral tissues and it was the major storage product in pancreas but was at very low levels in brain. These results suggest that the biosynthetic enzymes, GlcNAc transferase III (Narasimham, S. (1982) J. Biol. Chem. 257, 10235-10242) and IV (Gleeson, P.A., and Schachter, H. (1983) J. Biol. Chem. 258, 6162-6173), which are responsible for synthesis of these structures, have a generalized distribution with varying levels of expression in human viscera, moreover, transferase IV may have limited expression in neural tissue. The proposed structures for the branched-chain compounds are as follows. (formula; see text)

CT Check Tags: Comparative Study; Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
*Acetylglucosamine: AN, analysis
Brain Chemistry
Chemistry
*Glucosamine: AA, analogs & derivatives
Liver: AN, analysis
Magnetic Resonance Spectroscopy
*Oligosaccharides: AN, analysis
Pancreas: AN, analysis
***Sandhoff Disease: ME, metabolism**
Tissue Distribution

RN **3416-24-8 (Glucosamine)**; 7512-17-6 (Acetylglucosamine)

L122 ANSWER 36 OF 59 MEDLINE
ACCESSION NUMBER: 85152577 MEDLINE
DOCUMENT NUMBER: 85152577 PubMed ID: 3156697
TITLE: Late onset GM2 gangliosidosis: an alpha-locus genetic compound with near normal hexosaminidase activity.
AUTHOR: Charrow J; Inui K; Wenger D A
CONTRACT NUMBER: AM33170 (NIADDK)
HD08315 (NICHD)
SOURCE: CLINICAL GENETICS, (1985 Jan) 27 (1) 78-84.
Journal code: 0253664. ISSN: 0009-9163.
PUB. COUNTRY: Denmark
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198504
ENTRY DATE: Entered STN: 19900320
Last Updated on STN: 19970203
Entered Medline: 19850426

AB A non-Jewish child with late onset GM2 gangliosidosis is described. Tissues from the patient had near normal hexosaminidase A (hex A) activity using 4-methylumbelliferyl-2-acetamido-2-deoxy-beta-D-glucopyranoside

(MU-glcNAc) as substrate, and deficient activity when assayed with the 6-sulphate derivative of MU-glcNAc (MU-glcNAcS) or GM2 in the presence of activator. We present evidence that this patient is a genetic compound for different alpha-subunit mutations. The father's tissues have hex A activity in the heterozygote range when assayed with MU-glcNAcS, but normal activity using MU-glcNAc; the mother's tissues have activities toward both substrates in the heterozygote range. These results emphasize the pitfalls of using only MU-glcNAc for the diagnosis of unusual variants of GM2 gangliosidosis.

CT Check Tags: Case Report; Female; Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
Alleles
Child, Preschool
Glucosamine: AA, analogs & derivatives
Heterozygote
*Hexosaminidases: GE, genetics
Hymecromone: AA, analogs & derivatives
Mutation
Pedigree
Substrate Specificity
***Tay-Sachs Disease: EN, enzymology**
Tay-Sachs Disease: GE, genetics
beta-N-Acetylhexosaminidase

RN **3416-24-8 (Glucosamine)**; 37067-30-4 (4-methylumbelliferyl 2-acetamido-2-deoxy-beta-D-glucopyranoside); 90-33-5 (Hymecromone); 93751-71-4 (4-methylumbelliferyl-6-sulfo-2-acetamido-2-deoxy-beta-glucopyranoside)

L122 ANSWER 37 OF 59 MEDLINE
ACCESSION NUMBER: 85029011 MEDLINE
DOCUMENT NUMBER: 85029011 PubMed ID: 6436167
TITLE: Diagnosis of infantile and juvenile forms of GM2 gangliosidosis variant 0. Residual activities toward natural and different synthetic substrates.
AUTHOR: Kytzia H J; Hinrichs U; Sandhoff K
SOURCE: HUMAN GENETICS, (1984) 67 (4) 414-8.
Journal code: 7613873. ISSN: 0340-6717.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198412
ENTRY DATE: Entered STN: 19900320
Last Updated on STN: 19900320
Entered Medline: 19841207

AB p-Nitrophenyl-6-sulfo-2-acetamido-2-deoxy-beta-D-glucopyranoside, which is known to be a specific substrate for human hexosaminidase A, has recently been used successfully for diagnosis of variants B and B1 of GM2-gangliosidosis (Fuchs et al. 1983; Kytzia et al. 1983; Li et al. 1983). However, it is hydrolyzed by hexosaminidase S as well and is therefore not suitable for detection of patients with variant 0, who reach the normal range of activity toward this substrate. Assay of ganglioside GM2 cleaving activity in fibroblast extracts in the presence of the natural GM2 activator protein reveals residual hexosaminidase A activities of less than 2% of normal controls in two infantile and up to 7.5% in two juvenile patients with variant 0.

CT Check Tags: Human; Support, Non-U.S. Gov't
Cells, Cultured
Fibroblasts: EN, enzymology
G(M2) Ganglioside

***Gangliosidoses: DI, diagnosis**
Gangliosidoses: GE, genetics
Glucosamine: AA, analogs & derivatives
*Hexosaminidases: AN, analysis
Hymecromone: AA, analogs & derivatives
Isoelectric Focusing
Skin: PA, pathology
Substrate Specificity
Variation (Genetics)
RN 19600-01-2 (G(M2) Ganglioside); **3416-24-8 (Glucosamine)**; 37067-30-4 (4-methylumbelliferyl 2-acetamido-2-deoxy-beta-D-glucopyranoside); 90-33-5 (Hymecromone)

L122 ANSWER 38 OF 59 MEDLINE
ACCESSION NUMBER: 85143024 MEDLINE
DOCUMENT NUMBER: 85143024 PubMed ID: 6528335
TITLE: Role of lipid A in the production of tumor necrosis factor and differences in antitumor activity between tumor necrosis factor and lipopolysaccharide.
AUTHOR: Haranaka K; Satomi N; Sakurai A; Kunii O
SOURCE: TOHOKU JOURNAL OF EXPERIMENTAL MEDICINE, (1984 Dec) 144 (4) 385-96.
Journal code: 0417355. ISSN: 0040-8727.
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198503
ENTRY DATE: Entered STN: 19900320
Last Updated on STN: 19900320
Entered Medline: 19850328

AB The role of lipopolysaccharide (LPS) in the production of tumor necrosis factor (TNF) was examined. Alkaline treatment of LPS greatly reduced the TNF-producing activity of LPS, but TNF was produced when a large amount was injected. Free lipid A and lipid A-mouse serum albumin complex, which were prepared from the acid hydrolyzate, effectively induced TNF. However, the polysaccharide-rich fraction of the acid hydrolyzate was not capable of inducing TNF. Preincubation of LPS with polymixin B largely abrogated the TNF-producing activity of LPS. The differences in antitumor activity between TNF and LPS were also tested. TNF has a direct cytotoxicity against cancer cells in vitro but LPS does not. The activity of TNF was not inhibited by preincubation with polymixin B. Tumor necrosis in vivo was inhibited by preincubation of LPS with polymixin B but not by that of TNF. Galactosamine was found to induce susceptibility to the lethal effects of LPS, but did not influence the action of TNF. Lipid A is largely responsible for the TNF-inducing activity of LPS, but is not essential for the antitumor activity of TNF.

CT Check Tags: Animal; Female; Support, Non-U.S. Gov't
***Antineoplastic Agents: PD, pharmacology**
Galactosamine: PD, pharmacology
*Glycoproteins: BI, biosynthesis
*Growth Inhibitors: BI, biosynthesis
Hydrolysis
*Lipid A: PD, pharmacology
*Lipopolysaccharides: PD, pharmacology
Mammary Neoplasms, Experimental: DT, drug therapy
Mice
Mice, Inbred Strains
Polymyxin B: PD, pharmacology
Tumor Necrosis Factor

RN 1404-26-8 (Polymyxin B); **7535-00-4 (Galactosamine)**

L122 ANSWER 39 OF 59 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 84064855 EMBASE
DOCUMENT NUMBER: 1984064855
TITLE: The simple detection of neuraminic acid-containing urinary oligosaccharides in patients with glycoprotein storage diseases.
AUTHOR: Sewell A.C.
CORPORATE SOURCE: Department of Biochemistry, Royal Gwent Hospital, Newport NPT 2UB, United Kingdom
SOURCE: Journal of Inherited Metabolic Disease, (1983) 6/4 (153-157).
CODEN: JIMDDP
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal
FILE SEGMENT: 022 Human Genetics
029 Clinical Biochemistry
LANGUAGE: English
CT Medical Descriptors:
***gm1 gangliosidosis**
*mucolipidosis
*storage disease
human
etiology
congenital disorder
heredity
diagnosis
case report
Drug Descriptors:
*glycoprotein
*neuraminic acid
*oligosaccharide
RN (neuraminic acid) **114-04-5**

L122 ANSWER 40 OF 59 MEDLINE
ACCESSION NUMBER: 82208268 MEDLINE
DOCUMENT NUMBER: 82208268 PubMed ID: 6979385
TITLE: Antitumor activity of D-mannosamine in vitro: different sensitivities among human leukemia cell lines possessing T-cell properties.
AUTHOR: Onoda T; Morikawa S; Harada T; Suzuki Y; Inoue K; Nishigami K
SOURCE: CANCER RESEARCH, (1982 Jul) 42 (7) 2867-71.
Journal code: 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198208
ENTRY DATE: Entered STN: 19900317
Last Updated on STN: 19900317
Entered Medline: 19820814
AB D-Mannosamine is toxic to human malignant T-lymphoid cell lines derived from patients with T-cell leukemia. We observed heterogeneity of mannosamine susceptibility among those cell lines. The leukemic T-cell lines, subgrouped according to the degree of mannosamine inhibition on nucleic acid biosyntheses, were: Subgroup 1, HPB-MLT cells; Subgroup 2, CCRF-HSB-2 and HPB-ALL cells; and Subgroup 3, MOLT-4 cells. The most sensitive line, HPB-MLT, originated from the patient with adult T-cell

leukemia. The cytotoxicity of mannosamine was potentiated by a fatty acid, sodium oleate, at concentrations that were noncytolytic, and the interaction between the two drugs was synergistic. These results would suggest that mannosamine induces changes in the membrane structure of the leukemia cells. Thus, the primary target of the tumoricidal activity of mannosamine may also be the cellular membranes.

CT Check Tags: Human; Support, Non-U.S. Gov't
Adult
***Antineoplastic Agents: PD, pharmacology**
Cell Line
Cells, Cultured
Child
DNA, Neoplasm: BI, biosynthesis
*Hexosamines: PD, pharmacology
Hexosamines: TO, toxicity
*Leukemia: ME, metabolism
Lymphocyte Transformation: DE, drug effects
Middle Age
Monosaccharides: PD, pharmacology
Phytohemagglutinins: PD, pharmacology
*T-Lymphocytes

RN **2636-92-2 (mannosamine)**

L122 ANSWER 41 OF 59 MEDLINE
ACCESSION NUMBER: 81002382 MEDLINE
DOCUMENT NUMBER: 81002382 PubMed ID: 6773705
TITLE: Assay of the beta-glucosidase activity with natural labelled and artificial substrates in leukocytes from homozygotes and heterozygotes with the Norrbottnian type (Type 3) of Gaucher disease.
AUTHOR: Svennerholm L; Hakansson G; Dreborg S
SOURCE: CLINICA CHIMICA ACTA, (1980 Sep 25) 106 (2) 183-93. Journal code: 1302422. ISSN: 0009-8981.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198011
ENTRY DATE: Entered STN: 19900316
Last Updated on STN: 20000303
Entered Medline: 19801125

AB Leukocytes were isolated from 14 patients (7 males and 7 females) with Gaucher disease of the Norrbottnian type (Type 3), 32 obligate heterozygotes (16 males and 16 females) for this disease and 20 controls (10 males and 10 females). After collection, the cells were transported in dry ice to the laboratory, where they were assayed. The assays were repeated after the cells had been stored for 12 months. beta-Glucosidase activity was assayed with D-[glucose-U-14C]glucosylceramide at pH 5.8 with Cutscum-Na-cholate as a detergent and 4-methylumbelliferyl-beta-glucoside at pH 4.1 with Triton-Na-taurocholate as a detergent. The activities of two marker enzymes, 4-methylumbelliferyl-beta-galactosidase and N-acetyl-beta-glucosaminidase, were assayed in aliquots of the same leukocyte samples. The activity of beta-galactosidase remained constant during storage, N-acetyl-beta-glucosaminidase increased, while beta-glucosidase decreased as assayed with the natural as well as with the artificial substrate. beta-Glucosidase activity was significantly lower in the female than in male controls and heterozygotes. When assayed with natural substrate beta-glucosidase activity in leukocytes from the male patients was 6--12% of the control mean value and 10--15% in those from the female patients. The corresponding figures found when the artificial

substrate was used were 15--30% and 22--45%. The values for the heterozygotes were respectively 42--68% and 34--79% with the natural substrate, and 33--82% and 51--109% with the artificial substrate. No correlation was found between the age of the patient and the beta-glucosidase activity.

CT Check Tags: Female; Human; Male
Acetylglucosaminidase: ME, metabolism
Adolescence
Adult
Age Factors
Aged
Child
Child, Preschool
Drug Stability
Galactosides: ME, metabolism
***Gaucher Disease: EN, enzymology**
Gaucher Disease: GE, genetics
Glucosamine: AA, analogs & derivatives
Glucosamine: ME, metabolism
*Glucosidases: ME, metabolism
Glucosides: ME, metabolism
Glucosylceramidase: ME, metabolism
Heterozygote
Homozygote
Hymecromone: AA, analogs & derivatives
Hymecromone: ME, metabolism
*Leukocytes: EN, enzymology
Middle Age
Sex Factors
beta-Galactosidase: ME, metabolism
*beta-Glucosidase: ME, metabolism

RN 18997-57-4 (4-methylumbelliferyl glucoside); **3416-24-8 (Glucosamine)**; 37067-30-4 (4-methylumbelliferyl 2-acetamido-2-deoxy-beta-D-glucopyranoside); 6160-78-7 (4-methylumbelliferyl-galactopyranoside); 90-33-5 (Hymecromone)

L122 ANSWER 42 OF 59 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 80139148 MEDLINE
DOCUMENT NUMBER: 80139148 PubMed ID: 6244370
TITLE: Gangliosides containing glucosamine and galactosamine in transformed Tay-Sachs disease and normal human brain cell lines.
AUTHOR: Schneck L; Hoffman L M; Brooks S E; Amsterdam D
SOURCE: JOURNAL OF THE NEUROLOGICAL SCIENCES, (1980 Feb) 45 (1) 123-8.
Journal code: 0375403. ISSN: 0022-510X.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198005
ENTRY DATE: Entered STN: 19900315
Last Updated on STN: 19900315
Entered Medline: 19800514

AB Human SV-40 transformed brain cell lines derived from Tay-Sachs disease (TSD) and normal fetal cerebra were grown in culture and analyzed for their ganglioside content. Both the TSD and normal cells contained GM3, GM2, and a novel triheoxyl N-acetylglucosamine-containing ganglioside. In order to increase tumorigenicity, the cells were cloned on soft agar. The cloned cells still contained GM3, GM2, and the N-acetylglucosamine-

containing ganglioside. The per cent distribution of gangliosides in the TSD and normal SV-40 transformed cell lines was surprisingly similar despite the fact that the TSD transformed cells still lacked hexosaminidase A, the isoenzyme which is required to break down GM2.

CT Check Tags: Animal; Human
Cell Line
*Cell Transformation, Neoplastic: ME, metabolism
*Galactosamine: ME, metabolism
*Gangliosides: ME, metabolism
*Glucosamine: ME, metabolism
Simian virus 40
***Tay-Sachs Disease: ME, metabolism**
*Tumor Virus Infections: ME, metabolism

RN **3416-24-8 (Glucosamine); 7535-00-4 (Galactosamine)**

L122 ANSWER 43 OF 59 MEDLINE
ACCESSION NUMBER: 80068870 MEDLINE
DOCUMENT NUMBER: 80068870 PubMed ID: 41709
TITLE: Properties of multiple molecular forms of alpha-galactosidase and alpha-N-acetylgalactosaminidase from normal and Fabry leukocytes.
AUTHOR: Salvayre R; Maret A; Negre A; Douste-Blazy L
SOURCE: EUROPEAN JOURNAL OF BIOCHEMISTRY, (1979 Oct 15) 100 (2) 377-83.
Journal code: 0107600. ISSN: 0014-2956.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198002
ENTRY DATE: Entered STN: 19900315
Last Updated on STN: 19990129
Entered Medline: 19800215

CT Check Tags: Human
*Acetylgalactosamine: BL, blood
***Fabry Disease: EN, enzymology**
*Galactosamine: AA, analogs & derivatives
*Galactosidases: BL, blood
Glycosides: PD, pharmacology
Hydrogen-Ion Concentration
Isoelectric Focusing
*Isoenzymes: BL, blood
Kinetics
*Leukocytes: EN, enzymology
Osmolar Concentration
Structure-Activity Relationship
*alpha-Galactosidase: BL, blood

RN 31022-50-1 (Acetylgalactosamine); **7535-00-4 (Galactosamine)**

L122 ANSWER 44 OF 59 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1979:149859 BIOSIS
DOCUMENT NUMBER: BA67:29859
TITLE: GANGLIOSIDE GLYCOSYLATING ACTIVITY IN RAT BRAIN NEURONAL PERIKARYA FRACTION.
AUTHOR(S): MACCIONI H J F; DEFILPO S S; LANDA C A; CAPUTTO R
CORPORATE SOURCE: DEP. QUIM. BIOL., FAC. CIENC. QUIM., UNIV. NAC. CORDOBA, CIUDAD UNIV. CORDOBA, CORDOBA, ARGENT.
SOURCE: BIOCHEM J, (1978) 174 (3), 673-680.
CODEN: BIJOAK. ISSN: 0306-3275.
FILE SEGMENT: BA; OLD

LANGUAGE: English

AB Rat brain homogenate and the synaptosomal and neuronal perikarya fractions from 17 day old rats were compared for their activities in sialosylating endogenous gangliosides and transferring N-acetylneuraminic acid and galactose to several glycolipids in vitro. The sialosylation of endogenous gangliosides and the activities of sialosyltransferases acting either on lactosylceramide or hematoside as acceptors, as well as galactosyltransferase acting on **Tay-Sachs** ganglioside as acceptor, were between 3- and 12-fold higher in the neuronal perikarya fraction than in whole homogenate on a protein or ganglioside basis. The activities found in the synaptosomal fraction were negligible. No evidence was found to indicate that the low activities in this fraction were due to the presence of inhibitors of the transfer activities or to inaccessibility of the substrates to their respective enzymes. These findings and the time course of labeling of gangliosides of the neuronal perikarya and synaptosomes from rats that received an injection of N-[3H]acetylmannosamine indicate that the main cellular site of glycosylation of neuronal gangliosides is in the neuronal perikarya.

AB Rat brain homogenate and the synaptosomal and neuronal perikarya fractions from 17 day old rats were compared for their activities in sialosylating endogenous gangliosides and transferring N-acetylneuraminic acid and galactose to several glycolipids in vitro. The sialosylation of endogenous gangliosides and the activities of sialosyltransferases acting either on lactosylceramide or hematoside as acceptors, as well as galactosyltransferase acting on **Tay-Sachs** ganglioside as acceptor, were between 3- and 12-fold higher in the neuronal perikarya fraction than in whole homogenate on a protein or ganglioside basis. The activities found in the synaptosomal fraction were negligible. No evidence was found to indicate that the low activities in this fraction were due to the presence of inhibitors of the transfer activities or to inaccessibility of the substrates to their respective enzymes. These findings and the time course of labeling of gangliosides of the neuronal perikarya and synaptosomes from rats that received an injection of N-[3H]acetylmannosamine indicate that the main cellular site of glycosylation of neuronal gangliosides is in the neuronal perikarya.

IT Miscellaneous Descriptors
SIALOSYL TRANSFERASE GALACTOSYL TRANSFERASE N ACETYL NEURAMINIC-ACID GALACTOSE ACETYL MANNOSAMINE LACTOSYL CERAMIDE HEMATOSIDE **TAY SACHS** GANGLIOSIDE

RN 131-48-6 (N ACETYL NEURAMINIC-ACID)
4682-48-8 (LACTOSYL CERAMIDE)
9047-61-4 (TRANSFERASE)
59-23-4Q, 26566-61-0Q, 50855-33-9Q (GALACTOSE)
2636-92-2Q, 14307-02-9Q (MANNOSAMINE)
54827-14-4Q, 69345-49-9Q, 89678-50-2Q, 117465-88-0Q (HEMATOSIDE)

L122 ANSWER 45 OF 59 MEDLINE
ACCESSION NUMBER: 79023562 MEDLINE
DOCUMENT NUMBER: 79023562 PubMed ID: 699319
TITLE: Use of a chromogenic substrate for the diagnosis of Krabbe's disease, with special reference to its application in prenatal diagnosis.
AUTHOR: Besley G T; Bain A D
SOURCE: CLINICA CHIMICA ACTA, (1978 Sep 1) 88 (2) 229-36.
Journal code: 1302422. ISSN: 0009-8981.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197812

ENTRY DATE: Entered STN: 19900314
Last Updated on STN: 19900314
Entered Medline: 19781220

AB A chromogenic substrate, 2-hexadecanoylamino-4-nitrophenyl-beta-D-galactopyranoside, has recently been described for the diagnosis of Krabbe's disease. Hydrolysis of this substrate by extracts of cultured cells and tissues was compared with the activities of lactocerebrosidase I and non-specific beta-galactosidase. Under appropriate conditions, hydrolysis of the chromogenic analogue was markedly reduced in extracts of cultured amniotic fluid cells and skin fibroblasts derived from cases of Krabbe's disease. Activity was also markedly deficient in extracts of Krabbe's brain, although only a partial reduction was measured in liver extracts. Generally activities were higher in tissues of fetal origion. Unfortunately, the new analogue proved less specific and less sensitive than the natural substrates used to diagnose Krabbe's disease. Consequently, the analogue does not provide a satisfactory alternative substrate for the prenatal diagnosis of Krabbe's disease.

CT Check Tags: Female; Human; Male
Adolescence
Amniotic Fluid: CY, cytology
Brain: EN, enzymology
Cells, Cultured
Child, Preschool
*Enzyme Tests
Fibroblasts: EN, enzymology
*Galactosamine: AA, analogs & derivatives
*Galactosidases: ME, metabolism
Infant
Infant, Newborn
***Leukodystrophy, Globoid Cell: DI, diagnosis**
Liver: EN, enzymology
Pregnancy
*Prenatal Diagnosis
Skin: EN, enzymology

RN **7535-00-4 (Galactosamine)**

L122 ANSWER 46 OF 59 MEDLINE
ACCESSION NUMBER: 77087186 MEDLINE
DOCUMENT NUMBER: 77087186 PubMed ID: 827294
TITLE: The role of glycosidically bound mannose in the assimilation of beta-galactosidase by generalized gangliosidosis fibroblasts.
AUTHOR: Hieber V; Distler J; Myerowitz R; Schmickel R D; Jourdian G W
SOURCE: BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (1976 Dec 6) 73 (3) 710-7.
Journal code: 0372516. ISSN: 0006-291X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197702
ENTRY DATE: Entered STN: 19900313
Last Updated on STN: 20021218
Entered Medline: 19770224

CT Check Tags: Animal; Human; Male; Support, U.S. Gov't, P.H.S.
Aspergillus niger: EN, enzymology
Binding Sites
Biological Transport
Cattle

Chromatography, Affinity
Concanavalin A
Fibroblasts: DE, drug effects
Fibroblasts: ME, metabolism
Galactosidases: IP, isolation & purification
*Galactosidases: ME, metabolism
***Gangliosidoses: ME, metabolism**
Glucosamine: AN, analysis
Glucuronidase
Liver: EN, enzymology
*Mannose
Mannose: PD, pharmacology
Mannosidases
Mannosides: PD, pharmacology
Plant Lectins
Plants: EN, enzymology
Protein Binding
*Skin: ME, metabolism
Testis: EN, enzymology
RN 11028-71-0 (Concanavalin A); 31103-86-3 (Mannose); **3416-24-8 (Glucosamine)**

L122 ANSWER 47 OF 59 MEDLINE
ACCESSION NUMBER: 77002007 MEDLINE
DOCUMENT NUMBER: 77002007 PubMed ID: 822966
TITLE: Isolation of acidic glycopeptides from urine by means of anion-exchange resins. Application to some cases of glycosphingolipidosis or mucolipidosis.
AUTHOR: Calatroni A; Tira M E
SOURCE: CLINICA CHIMICA ACTA, (1976 Sep 6) 71 (2) 137-41.
Journal code: 1302422. ISSN: 0009-8981.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197612
ENTRY DATE: Entered STN: 19900313
Last Updated on STN: 19900313
Entered Medline: 19761201
AB An acidic fraction containing aminosugar was isolated by means of Dowex 1 from normal human urine which had previously been filtered through Ecteolacellulose. After purification, the fraction was shown to be composed of peptides and carbohydrates in comparable amounts. Threonine, serine and dicarboxylic acids were the principal amino acids. The carbohydrate moiety was mainly composed of galactose and glucosamine in the approximate ratio 3 : 1, together with smaller amounts of fucose, sialic acid, galactosamine and mannose. The presence of an O-glycosidic bond to threonine was shown by alkali treatment in reducing conditions. The fraction is probably a mixture of acidic glycopeptides. Fractions showing similar characteristics were isolated from urine of patients with Niemann-Pick disease, Gaucher's disease, I-cell disease, Ehlers-Danlos syndrome. Slight differences from the normal were found in the composition of the fraction isolated from GM1-gangliosidosis type 1.
CT Check Tags: Comparative Study; Human; Male
Amino Acids: AN, analysis
Chromatography, Ion Exchange
Galactosamine: AN, analysis
Glucosamine: AN, analysis
*Glycopeptides: UR, urine
Hexoses: AN, analysis

Sialic Acids: AN, analysis
***Sphingolipidoses: UR, urine**
RN **3416-24-8 (Glucosamine); 7535-00-4 (Galactosamine)**

L122 ANSWER 48 OF 59 MEDLINE
ACCESSION NUMBER: 76228355 MEDLINE
DOCUMENT NUMBER: 76228355 PubMed ID: 820167
TITLE: Storage and excretion of oligosaccharides and glycopeptides in the gangliosidoses.
AUTHOR: Wolfe L S; Ng Kin Kin N M
SOURCE: ADVANCES IN EXPERIMENTAL MEDICINE AND BIOLOGY, (1976) 68 15-29.
Journal code: 0121103. ISSN: 0065-2598.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197608
ENTRY DATE: Entered STN: 19900313
Last Updated on STN: 19900313
Entered Medline: 19760823
CT Check Tags: Human
Galactose: AN, analysis
***Gangliosidoses: ME, metabolism**
Glucosamine: AN, analysis
*Glycopeptides: ME, metabolism
Infant
Lipoidosis: ME, metabolism
Liver: ME, metabolism
Mannose: AN, analysis
Molecular Conformation
Molecular Weight
*Oligosaccharides: ME, metabolism
RN 26566-61-0 (Galactose); 31103-86-3 (Mannose); **3416-24-8 (Glucosamine)**

L122 ANSWER 49 OF 59 MEDLINE
ACCESSION NUMBER: 75082559 MEDLINE
DOCUMENT NUMBER: 75082559 PubMed ID: 4280528
TITLE: Steroid hexosaminidase activity in Tay-Sachs and Sandhoff-Jatzkewitz diseases.
AUTHOR: Tomasi L G; Fukushima D K; Kolodny E H
SOURCE: NEUROLOGY, (1974 Dec) 24 (12) 1158-65.
Journal code: 0401060. ISSN: 0028-3878.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197504
ENTRY DATE: Entered STN: 19900310
Last Updated on STN: 19970203
Entered Medline: 19750419
CT Check Tags: Comparative Study; Human
Carbon Radioisotopes
Catalysis
Child, Preschool
*Galactosidases: AN, analysis
*Gangliosides
Glucosamine: AA, analogs & derivatives
Glucosamine: UR, urine

*Glucosaminidase: AN, analysis
Infant
Kinetics
*Lipoidosis: EN, enzymology
Lipoidosis: UR, urine
*Liver: EN, enzymology
Prasterone
***Sphingolipidoses: EN, enzymology**
Sphingolipidoses: UR, urine
Testosterone
RN **3416-24-8 (Glucosamine)**; 53-43-0 (Prasterone); 57-85-2 (Testosterone)

L122 ANSWER 50 OF 59 MEDLINE
ACCESSION NUMBER: 75009193 MEDLINE
DOCUMENT NUMBER: 75009193 PubMed ID: 4213050
TITLE: Editorial: Synthetic defect in ganglioside synthesis.
AUTHOR: O'Brien J S
SOURCE: NEW ENGLAND JOURNAL OF MEDICINE, (1974 Oct 31) 291 (18) 975-6.
Journal code: 0255562. ISSN: 0028-4793.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 197412
ENTRY DATE: Entered STN: 19900310
Last Updated on STN: 19900310
Entered Medline: 19741217
CT Check Tags: Human; Male
Autopsy
Brain: ME, metabolism
Galactosamine
*Gangliosides: BI, biosynthesis
Gangliosides: ME, metabolism
Glycolipids
Hexosyltransferases: DF, deficiency
Infant
Liver: ME, metabolism
Sphingolipidoses: EN, enzymology
***Sphingolipidoses: ME, metabolism**
Sphingolipidoses: PA, pathology
Uridine Diphosphate Sugars
RN **7535-00-4 (Galactosamine)**

L122 ANSWER 51 OF 59 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1975:109287 BIOSIS
DOCUMENT NUMBER: BA59:9287
TITLE: SANDHOFF DISEASE DEFECTIVE GLYCOSAMINO GLYCAN CATABOLISM IN CULTURED FIBROBLASTS AND ITS CORRECTION BY BETA-N ACETYL HEXOSAMINIDASE.
AUTHOR(S): CANTZ M; KRESSE H
SOURCE: EUR J BIOCHEM, (1974) 47 (3), 581-590.
CODEN: EJBCAI. ISSN: 0014-2956.
FILE SEGMENT: BA; OLD
LANGUAGE: Unavailable
IT Miscellaneous Descriptors
HUMAN URINE CHONDROITIN SULFATE DERMATAN SULFATE **TAY SACHS** ISOZYME A EC-3.2.1.52 ISOZYME B HYALURONIC-ACID CARBON-14 GLUCOSAMINE

RN **3416-24-8** (GLUCOSAMINE)
9004-61-9 (HYALURONIC-ACID)
9007-28-7 (CHONDROITIN SULFATE)
9027-52-5 (BETA-N ACETYL HEXOSAMINIDASE)
9027-52-5 (EC-3.2.1.52)
14762-75-5 (CARBON-14)
24967-94-0 (DERMATAN SULFATE)
3416-24-8 (GLUCOSAMINE)
9004-61-9 (HYALURONIC-ACID)
9007-28-7 (CHONDROITIN SULFATE)
9027-52-5 (BETA-N ACETYL HEXOSAMINIDASE)
9027-52-5 (EC-3.2.1.52)
14762-75-5 (CARBON-14)
24967-94-0 (DERMATAN SULFATE)

L122 ANSWER 52 OF 59 MEDLINE
ACCESSION NUMBER: 74276476 MEDLINE
DOCUMENT NUMBER: 74276476 PubMed ID: 4210440
TITLE: [Juvenile GM2 gangliosidosis with altered substrate specificity of hexosaminidase A (author's transl)]. Juvenile GM2-gangliosidose mit veranderter Substratspezifitat der Hexosaminidase A.
AUTHOR: Zerfowski J; Sandhoff K
SOURCE: ACTA NEUROPATHOLOGICA, (1974 Mar 26) 27 (3) 225-32. Journal code: 0412041. ISSN: 0001-6322.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197409
ENTRY DATE: Entered STN: 19900310
Last Updated on STN: 19900310
Entered Medline: 19740917
CT Check Tags: Female; Human
Adolescence
Ceramides
Galactosamine
Galactosaminidase: ME, metabolism
*Hexosaminidases: ME, metabolism
Hydrogen-Ion Concentration
*Liver: EN, enzymology
Neuraminic Acids
***Sphingolipidoses: EN, enzymology**
RN **7535-00-4 (Galactosamine)**

L122 ANSWER 53 OF 59 MEDLINE
ACCESSION NUMBER: 75100885 MEDLINE
DOCUMENT NUMBER: 75100885 PubMed ID: 4217436
TITLE: Chemotherapy of malignant mesothelioma.
AUTHOR: Gerner R E; Moore G E
SOURCE: ONCOLOGY, (1974) 30 (2) 152-5. Journal code: 0135054. ISSN: 0030-2414.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197505
ENTRY DATE: Entered STN: 19900310
Last Updated on STN: 19900310
Entered Medline: 19750506

CT Check Tags: Female; Human; Male
Adult
Aged
***Antineoplastic Agents: TU, therapeutic use**
Fluorouracil: TU, therapeutic use
Glucosamine: TU, therapeutic use
*Mesothelioma: DT, drug therapy
Middle Age
*Peritoneal Neoplasms: DT, drug therapy
*Pleural Neoplasms: DT, drug therapy
Thiotepa: TU, therapeutic use
RN **3416-24-8 (Glucosamine)**; 51-21-8 (Fluorouracil); 52-24-4 (Thiotepa)

L122 ANSWER 54 OF 59 MEDLINE
ACCESSION NUMBER: 74045510 MEDLINE
DOCUMENT NUMBER: 74045510 PubMed ID: 4271344
TITLE: Altered levels of tissue glycoproteins, gangliosides, glycosaminoglycans and lipids in Niemann-Pick's disease.
AUTHOR: Brunngraber E G; Berra B; Zambotti V
SOURCE: CLINICA CHIMICA ACTA, (1973 Oct 12) 48 (2) 173-81. Journal code: 1302422. ISSN: 0009-8981.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197401
ENTRY DATE: Entered STN: 19900310
Last Updated on STN: 20000303
Entered Medline: 19740131
CT Check Tags: Human
Brain Chemistry
Cell Membrane: AN, analysis
Cellulose
Child, Preschool
Cholesterol: AN, analysis
Chromatography, Gel
Chromatography, Ion Exchange
Chromatography, Paper
Chromatography, Thin Layer
Electrophoresis
*Gangliosides: AN, analysis
Glucosamine: AN, analysis
*Glycoproteins: AN, analysis
*Glycosaminoglycans: AN, analysis
Hexoses: AN, analysis
Infant
*Lipids: AN, analysis
Liver: AN, analysis
Neuraminic Acids: AN, analysis
***Niemann-Pick Diseases: ME, metabolism**
Niemann-Pick Diseases: PA, pathology
Phospholipids: AN, analysis
Sphingomyelins: AN, analysis
RN **3416-24-8 (Glucosamine)**; 57-88-5 (Cholesterol); 9004-34-6 (Cellulose)

L122 ANSWER 55 OF 59 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1973:91346 BIOSIS
DOCUMENT NUMBER: BR09:91346

TITLE: INCORPORATION OF SELECTED ISOTOPES INTO LIPIDS OF HUMANS WITH CEREBRAL LIPIDOSIS D GLUCOSAMINE 1 CARBON-14.
AUTHOR(S): BURTON R M; HANDA S; HOWARD R E; VIETTI T; RAGAB A
SOURCE: J. Am. Oil Chem. Soc., (1973) 50 (2), 86A.
CODEN: JAOCA7. ISSN: 0003-021X.
DOCUMENT TYPE: Conference
FILE SEGMENT: BR; OLD
LANGUAGE: Unavailable
IT Miscellaneous Descriptors
ABSTRACT **TAY SACHS** DISEASE NIEMANN PICKS DISEASE
RN **3416-24-8** (D GLUCOSAMINE)
14762-75-5 (CARBON-14)

L122 ANSWER 56 OF 59 MEDLINE
ACCESSION NUMBER: 73087805 MEDLINE
DOCUMENT NUMBER: 73087805 PubMed ID: 4675278
TITLE: Antineoplastic drug activity in the mitotic cycle--effects of six agents on macromolecular synthesis in synchronous mammalian leukemic cells.
AUTHOR: Bosmann H B
SOURCE: BIOCHEMICAL PHARMACOLOGY, (1972 Jul 15) 21 (14) 1977-88.
Journal code: 0101032. ISSN: 0006-2952.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197303
ENTRY DATE: Entered STN: 19900310
Last Updated on STN: 19900310
Entered Medline: 19730323
CT Check Tags: Animal
***Antineoplastic Agents: PD, pharmacology**
Asparaginase: PD, pharmacology
Azaserine: PD, pharmacology
Camptothecin: PD, pharmacology
Culture Media
*DNA, Neoplasm: BI, biosynthesis
Ethidium: PD, pharmacology
Fucose: ME, metabolism
Glucosamine: PD, pharmacology
*Glycoproteins: BI, biosynthesis
Hydroxyurea: PD, pharmacology
Leucine: ME, metabolism
*Leukemia, Experimental: ME, metabolism
Mice
*Mitosis: DE, drug effects
Neoplasm Proteins: AN, analysis
*Neoplasm Proteins: BI, biosynthesis
*RNA, Neoplasm: BI, biosynthesis
Thymidine: ME, metabolism
Time Factors
Tritium
Uridine: ME, metabolism
RN 10028-17-8 (Tritium); 115-02-6 (Azaserine); 127-07-1 (Hydroxyurea); **3416-24-8 (Glucosamine)**; 3546-21-2 (Ethidium); 3713-31-3 (Fucose); 50-89-5 (Thymidine); 58-96-8 (Uridine); 61-90-5 (Leucine); 7689-03-4 (Camptothecin)

L122 ANSWER 57 OF 59 MEDLINE
ACCESSION NUMBER: 71059418 MEDLINE

DOCUMENT NUMBER: 71059418 PubMed ID: 4992220
TITLE: Camptothecin inhibits macromolecular synthesis in mammalian cells but not in isolated mitochondria of E. coli.
AUTHOR: Bosmann H B
SOURCE: BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (1970 Dec 24) 41 (6) 1412-20.
Journal code: 0372516. ISSN: 0006-291X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197102
ENTRY DATE: Entered STN: 19900101
Last Updated on STN: 19970203
Entered Medline: 19710204

CT Check Tags: Animal
*Alkaloids: PD, pharmacology
*Antibiotics: PD, pharmacology
***Antineoplastic Agents: PD, pharmacology**
Brain: CY, cytology
Brain: ME, metabolism
Carbon Isotopes
Cell Line
*DNA: BI, biosynthesis
DNA, Bacterial: BI, biosynthesis
Edetic Acid
*Escherichia coli: ME, metabolism
Glucosamine: ME, metabolism
Glycoproteins: BI, biosynthesis
Hela Cells: ME, metabolism
Leucine: ME, metabolism
Lipopolysaccharides: BI, biosynthesis
Lymphoma
Metabolism: DE, drug effects
Mice
*Mitochondria: ME, metabolism
Mitochondria, Liver: ME, metabolism
*Proteins: BI, biosynthesis
*RNA: BI, biosynthesis
Rats
Thymidine: ME, metabolism
Tritium
Uridine: ME, metabolism

RN 10028-17-8 (Tritium); **3416-24-8 (Glucosamine)**; 50-89-5 (Thymidine); 58-96-8 (Uridine); 60-00-4 (Edetic Acid); 61-90-5 (Leucine); 63231-63-0 (RNA); 9007-49-2 (DNA)

L122 ANSWER 58 OF 59 MEDLINE
ACCESSION NUMBER: 71238455 MEDLINE
DOCUMENT NUMBER: 71238455 PubMed ID: 4326600
TITLE: [Biochemical studies of the action of immunosuppressive substances].
Biochemische Untersuchungen zur Wirkungsweise immunsuppressiver Substanzen.
AUTHOR: Greiling H
SOURCE: VERHANDLUNGEN DER DEUTSCHEN GESELLSCHAFT FUR RHEUMATOLOGIE, (1969) 1 370-7.
Journal code: 7507680. ISSN: 0070-4121.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197108
ENTRY DATE: Entered STN: 19900101
Last Updated on STN: 19980206
Entered Medline: 19710830
CT Check Tags: Animal; Human; Male
Adult
***Antineoplastic Agents: PD, pharmacology**
Arthritis, Rheumatoid: BL, blood
Arthritis, Rheumatoid: DT, drug therapy
Arthritis, Rheumatoid: EN, enzymology
Arthritis, Rheumatoid: ME, metabolism
Carbon Isotopes
Cattle
Connective Tissue: DE, drug effects
Connective Tissue: ME, metabolism
Cornea
Fructose-Bisphosphate Aldolase: ME, metabolism
Glucosamine: ME, metabolism
Glycosaminoglycans: BI, biosynthesis
Glycoside Hydrolases: ME, metabolism
Immunoglobulins: BI, biosynthesis
*Immunosuppressive Agents: PD, pharmacology
Middle Age
Models, Biological
Peptide Synthesis
Phenylbutazone: PD, pharmacology
Phosphoric Monoester Hydrolases: ME, metabolism
*Plants, Medicinal
*Plants, Toxic
Podophyllin: PD, pharmacology
Podophyllin: TU, therapeutic use
*Podophyllum: PD, pharmacology
*Puromycin: PD, pharmacology
Serine: ME, metabolism
Sulfates: ME, metabolism
Sulfur Isotopes
Synovial Fluid: EN, enzymology
RN **3416-24-8 (Glucosamine)**; 50-33-9 (Phenylbutazone); 53-79-2 (Puromycin); 56-45-1 (Serine); 9000-55-9 (Podophyllin)

L122 ANSWER 59 OF 59 MEDLINE
ACCESSION NUMBER: 69031214 MEDLINE
DOCUMENT NUMBER: 69031214 PubMed ID: 5688474
TITLE: Incorporation of selected isotopes into lipids of humans with cerebral lipidoses: studies on D-glucosamine-1-14C.
AUTHOR: Burton R; Handa S; Howard R E; Vietti T
SOURCE: PATHOLOGIA EUROPAEA, (1968) 3 (2) 424-30.
Journal code: 0062702. ISSN: 0031-2967.
PUB. COUNTRY: Belgium
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 196812
ENTRY DATE: Entered STN: 19900101
Last Updated on STN: 20000303
Entered Medline: 19681230
CT Check Tags: Female; Human
*Brain: ME, metabolism

Carbon Isotopes
Child
Child, Preschool
*Gangliosides: BI, biosynthesis
*Glucosamine: ME, metabolism
*Lipids: ME, metabolism
*Lipoidosis: ME, metabolism
***Niemann-Pick Diseases: ME, metabolism**
*Sphingomyelins: ME, metabolism
RN **3416-24-8 (Glucosamine)**